The American Journal of Surgery

EDITED BY THURSTON SCOTT WELTON, M.D.

Symposium

ACUTE SURGICAL CONDITIONS OF THE ABDOMEN

Edited by

ARNOLD S. JACKSON, M.D.

FIFTY-FIFTH YEAR OF CONTINUOUS PUBLICATION

December 1946

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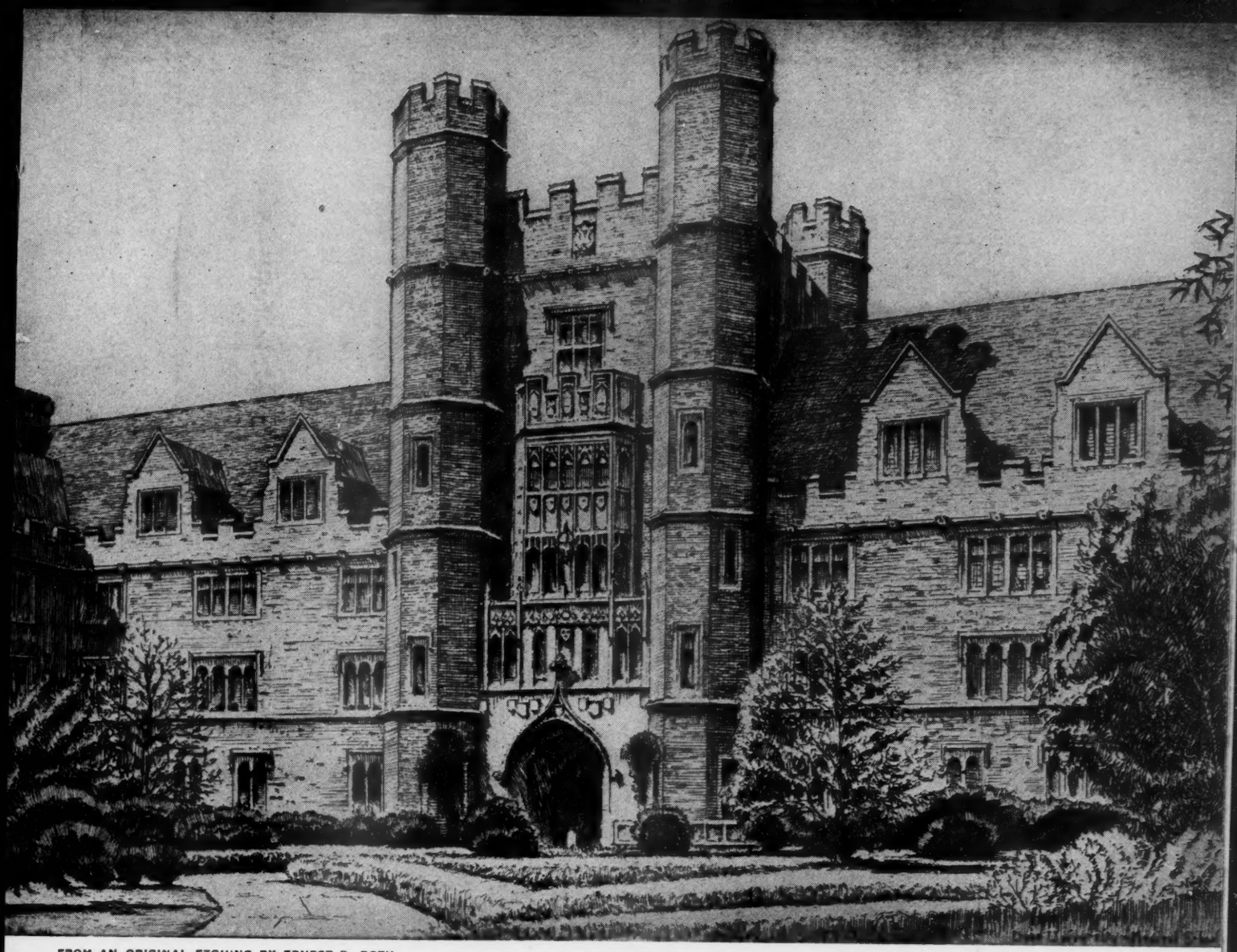

FROM AN ORIGINAL ETCHING BY ERNEST D. ROTH

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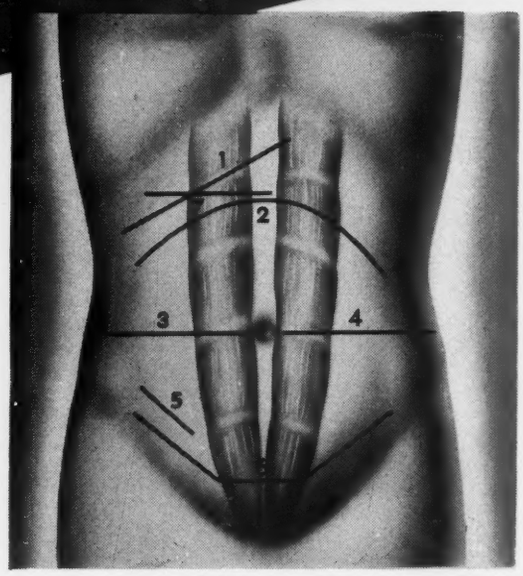
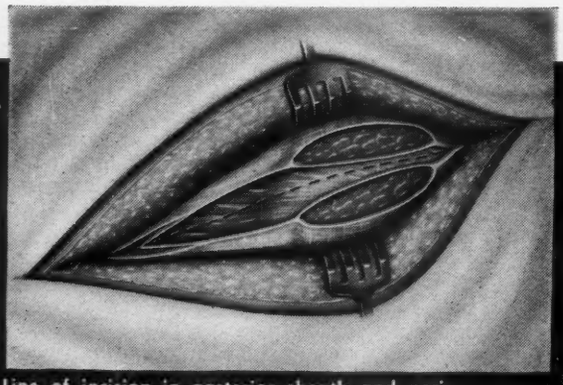
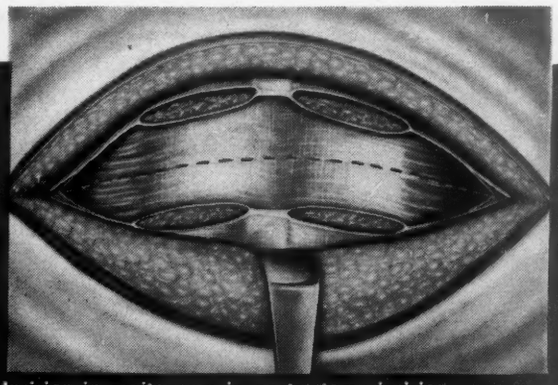
A resumé of the principles involved, together with some pictures describing the incisions, are shown on these two pages. This series covers the principles as they relate to the abdominal section of the body.



1. For gall bladder, common duct, head of pancreas.
2. Resection of stomach, transverse colon.
3. Resection of ascending colon, cecum, right sided lesions.
4. Descending colon, spleen and left sided lesions
5. Appendectomy.
6. Pelvic surgery, resection of sigmoid.
7. Small exploratory transverse incision.



Line of incision in posterior sheath and peritoneum in gall bladder incision.



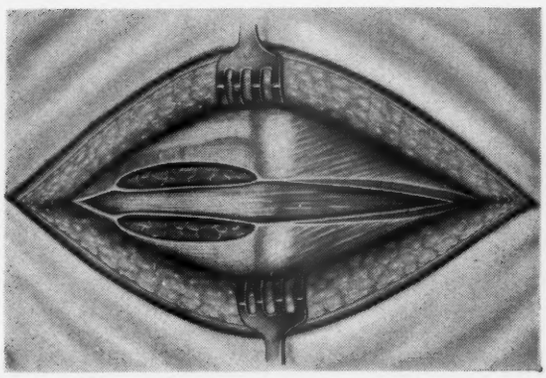
Incision in peritoneum in gastrectomy incision.

ADVANTAGES OF TRANSVERSE INCISIONS

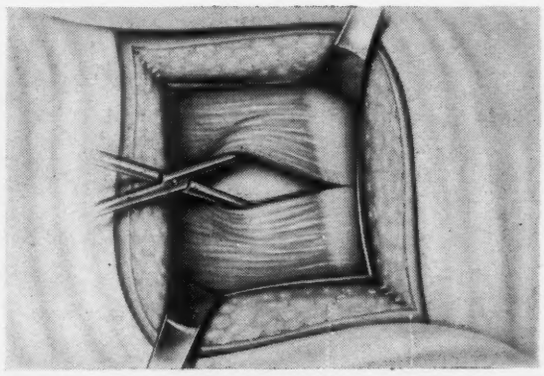
SUTURES hold more securely if the connective tissue layers within the wound are approximated in such a manner that the sutures must pull against the fibers within them. Sutures pull against the fibers only when the connective tissues are cut parallel to the direction of the fibers. In the anterior abdominal wall, this is usually best accomplished through a transverse incision. As a result, sutured transverse incisions are stronger than sutured vertical incisions.

In the past, transverse abdominal incisions did not find a wide acceptance because they necessitated cutting across muscle. Attempts were made to suture the muscles and the sutures not only held poorly, but they almost invariably caused necrosis.

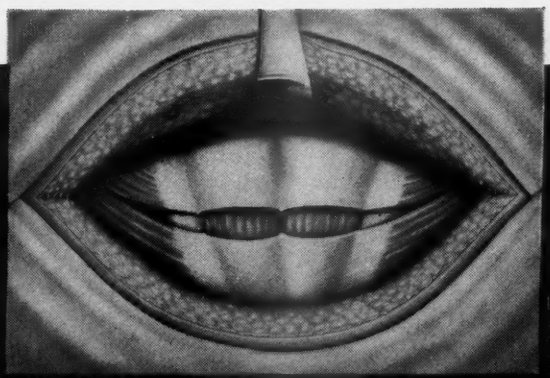
In the new technic, no attempt is made to suture muscles—only the surrounding sheath. Necrosis is thereby avoided. Some approximation of muscle is obtained by suturing their sheath, and further approximation is accomplished by keeping the patient in a semi-reclining position with knees partially reflexed.



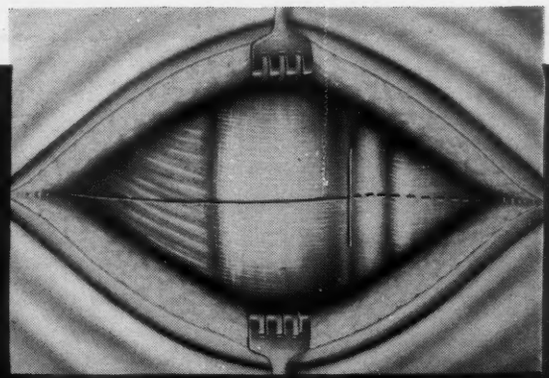
Cut rectus muscle in the incision for resecting the descending colon.



Separating muscle fibers in McBurney incision.



Cutting the fascia of the anterior sheath in lower abdominal transverse incision.



Incision in anterior rectus sheath with possible extension.

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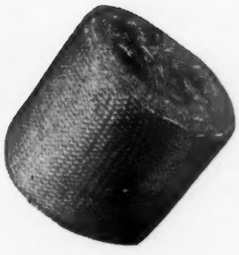

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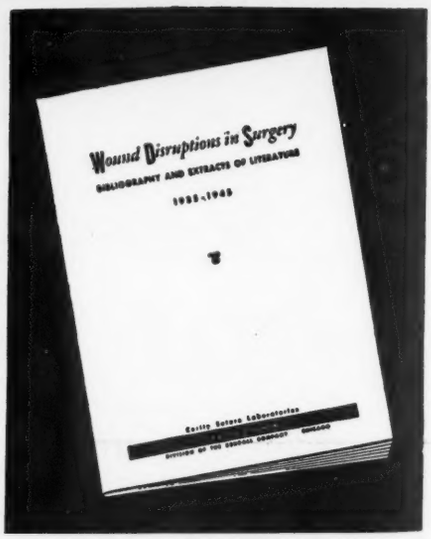

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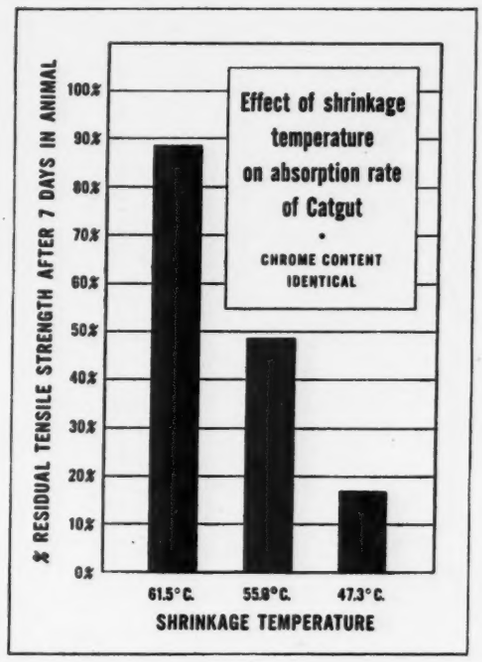

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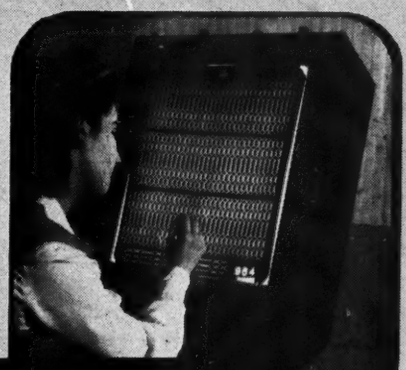

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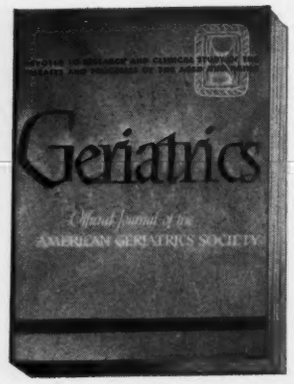

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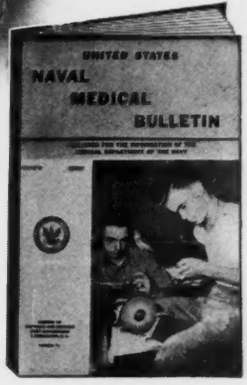
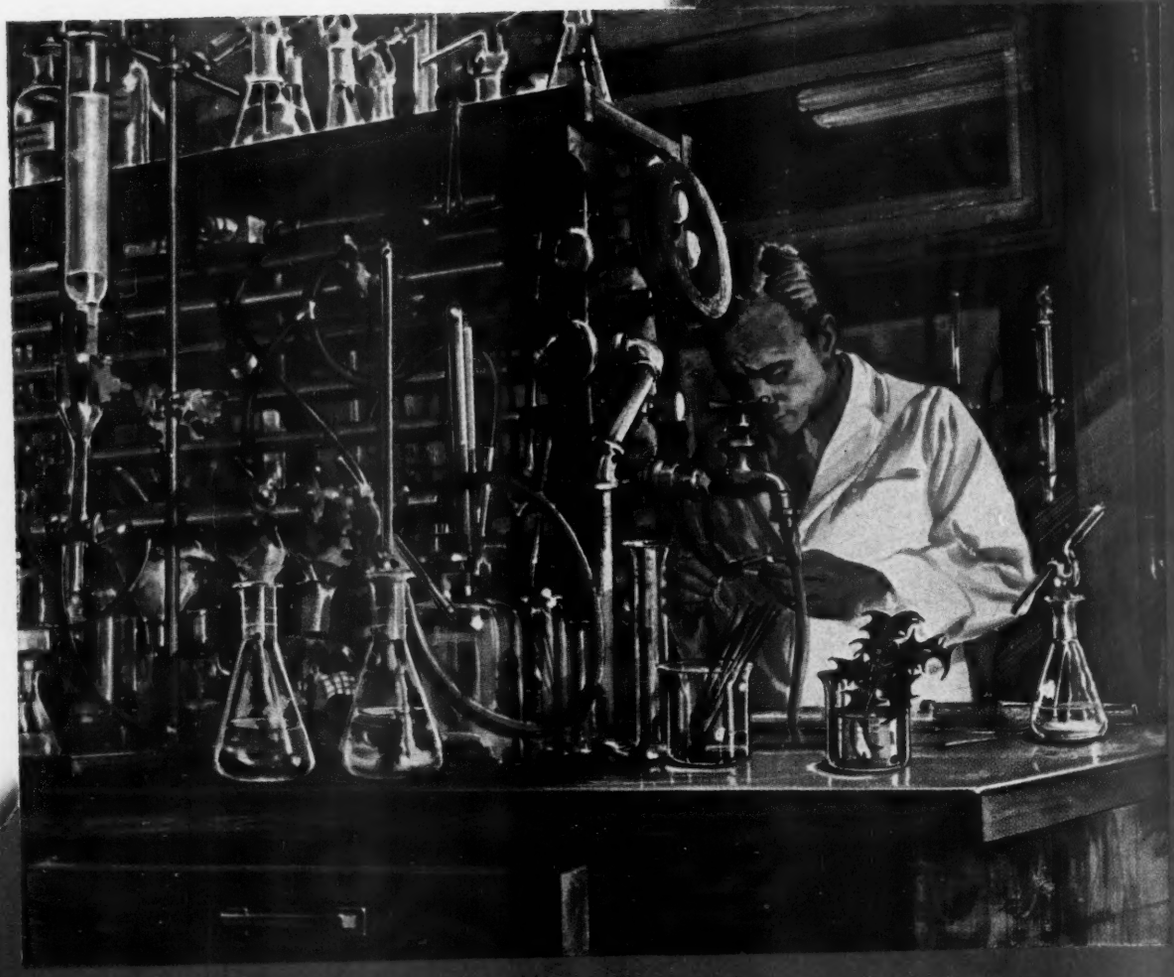


1. Gunther, L., U. S. Naval Med. Bull., 44:300, 1945.



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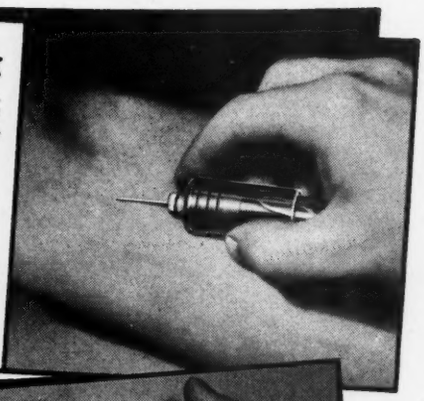

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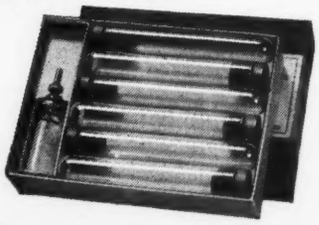

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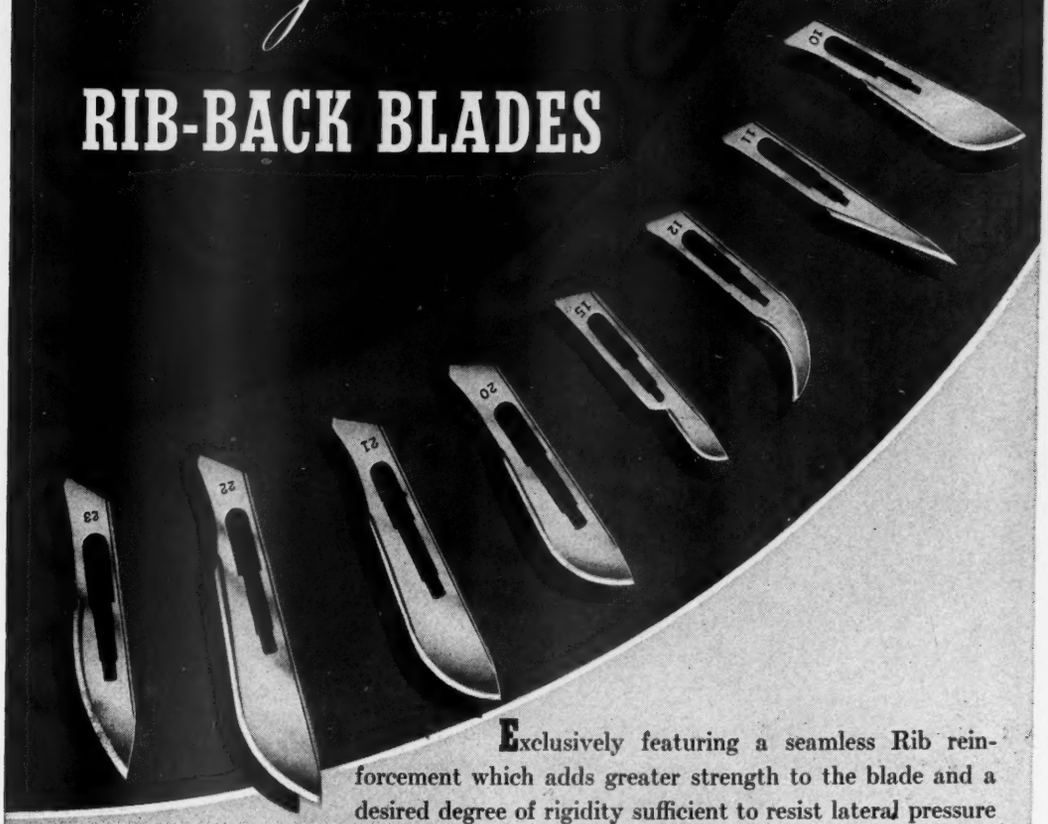

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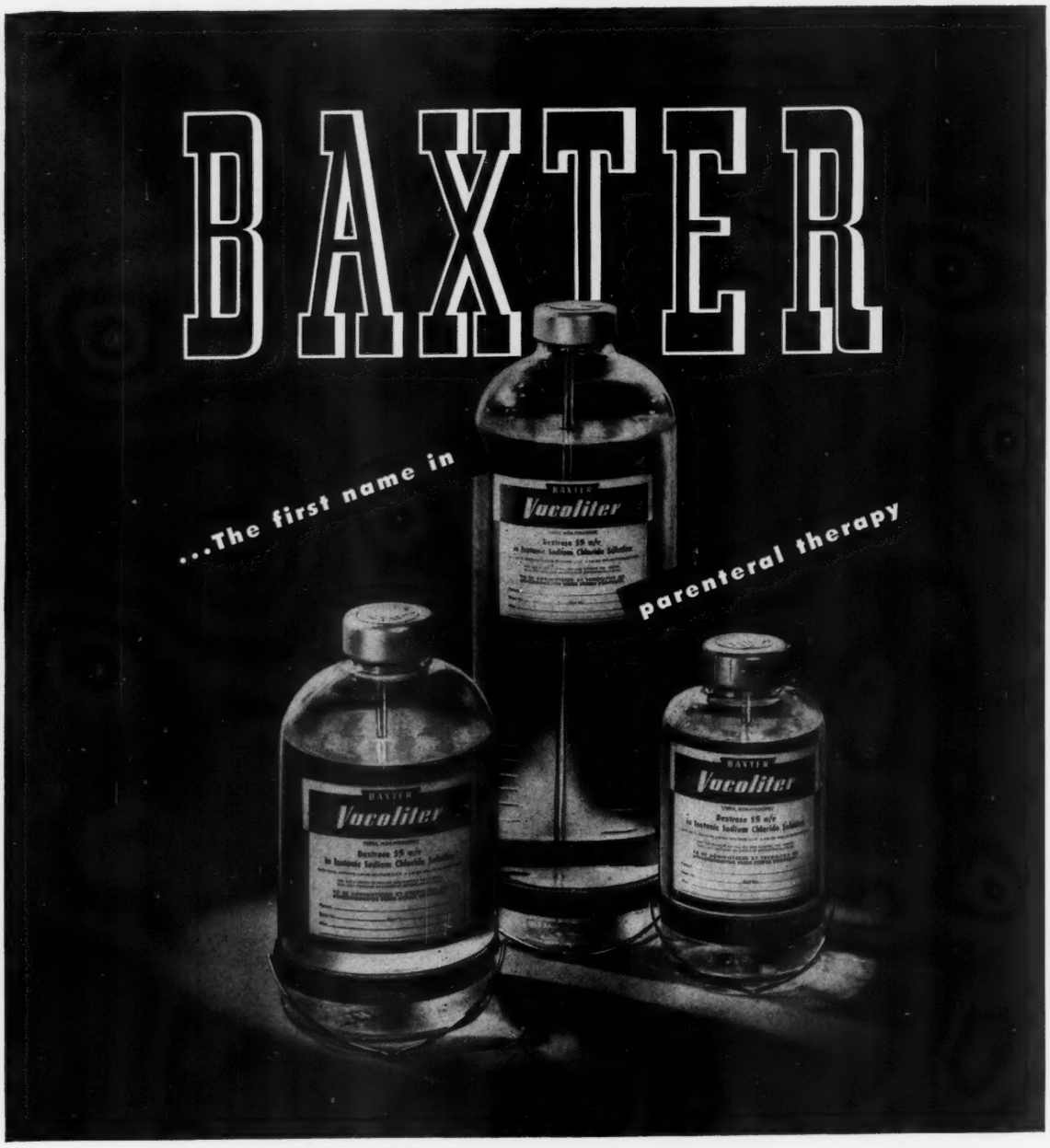

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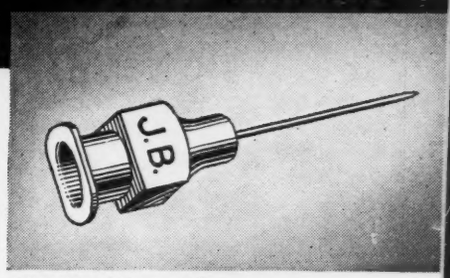

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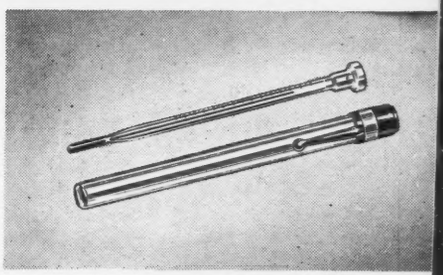

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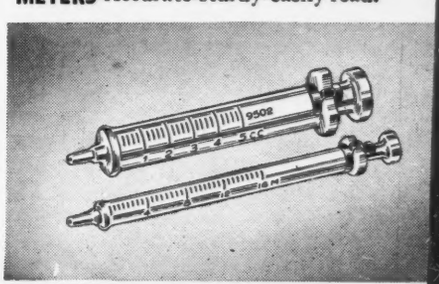

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Such an improved dressing must allow proper drainage and granulation of the wound so that an early split-skin graft can be applied if desired. It should be non-irritating to the skin and should not act as a foreign body if left embedded in human tissues. It should prevent adherence of granulations and prevent exuberant granulation. It should not cause pain or excessive bleeding on removal, and should be simple to prepare and use.

United States Army Signal Corps Photo.

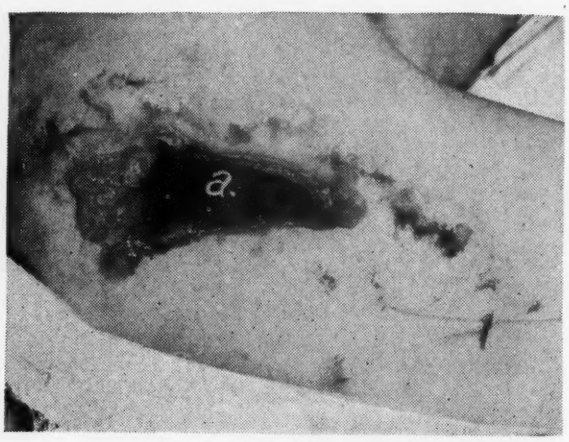
Shoulder wound, three weeks after saucerization of the cavity. (a) Fiberglas Cloth may be seen on the wound surfaces. (b) No granulations are growing through the meshes.

After consideration of these factors, Fiberglas Cloth was tried experimentally at the Percy Jones General Hospital, Battle Creek, Mich., in thirty-five cases where ordinarily petrolatum-impregnated gauze would have been used.

METHOD OF APPLICATION

In applying the dressing at the time of surgery, a square of glass cloth is cut large enough to cover all surfaces of the wound and to project over the skin margin. The center of the invaginated glass cloth should be packed with dry cotton gauze to hold open the edges of the wound and to act as a wick to remove exudate. A pressure bandage or padded cast, as indicated, is placed over such a dressing.

These Fiberglas dressings were left in the wound for varying periods of time. The report in the *Journal of Bone and Joint Surgery** says, "contra-indications have not been observed". Fiberglas Cloth was also tried as the first layer of a wet dressing for a split-skin graft. By such experimentation, the uses of Fiberglas in medicine are being established.



United States Army Signal Corps Photo.

The same wound (a) after removal of Fiberglas Cloth, showing clean granulations and no bleeding or thick exudate.

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*Vol. 28, No. 1, pp. 180-181, January, 1946.

Some Other Uses of Fiberglas in Medicine

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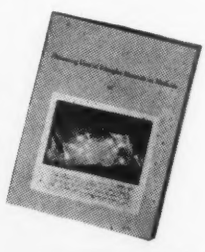

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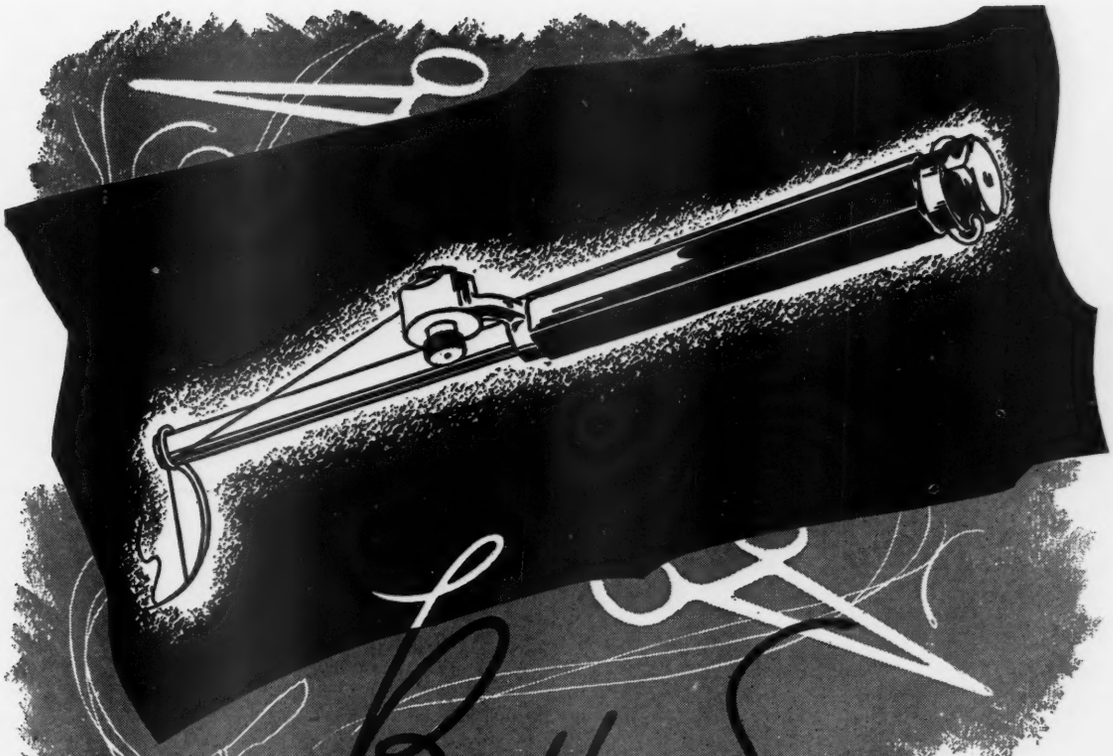

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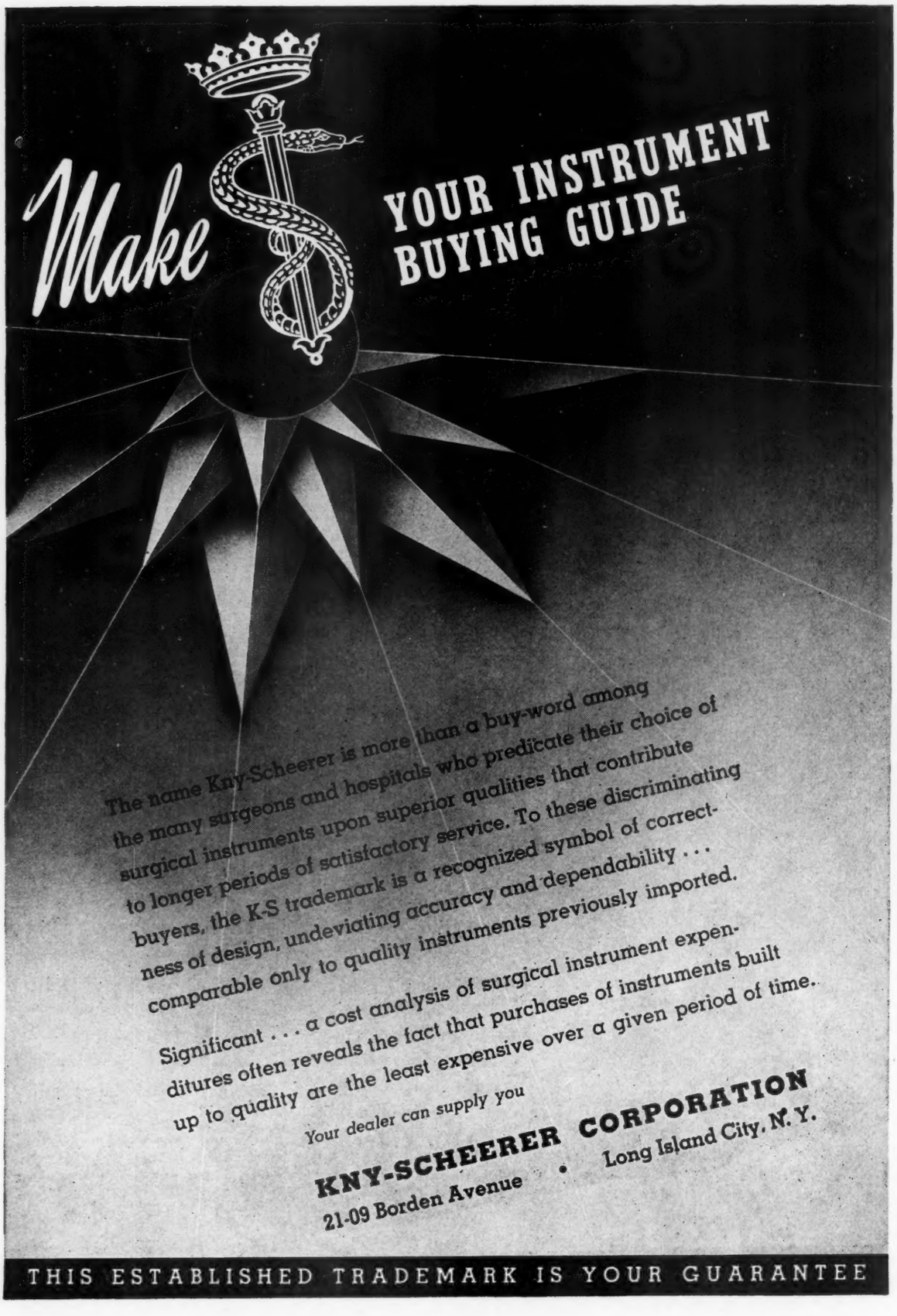
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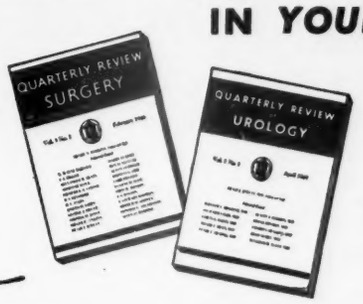

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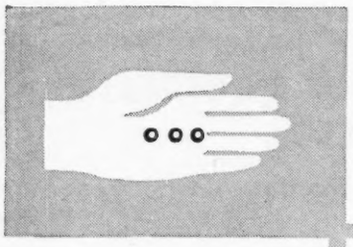

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2. Editorial: J.A.M.A. 127:1056, 1945.
3. Thompson: Biochem. J. 34:959, 1940.

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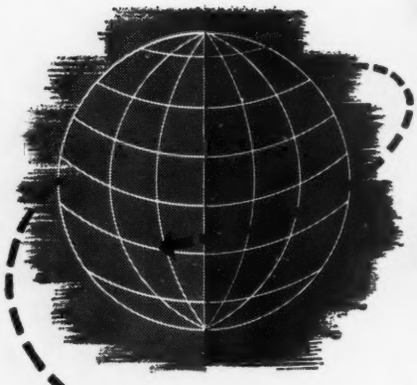

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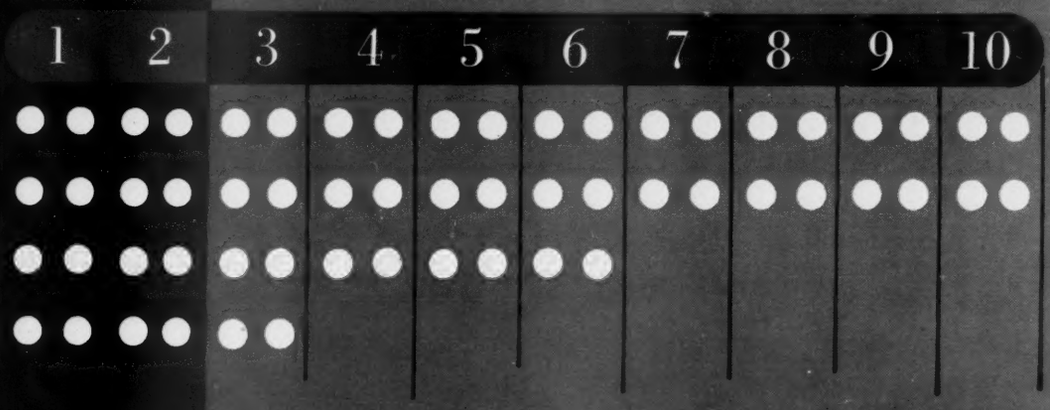

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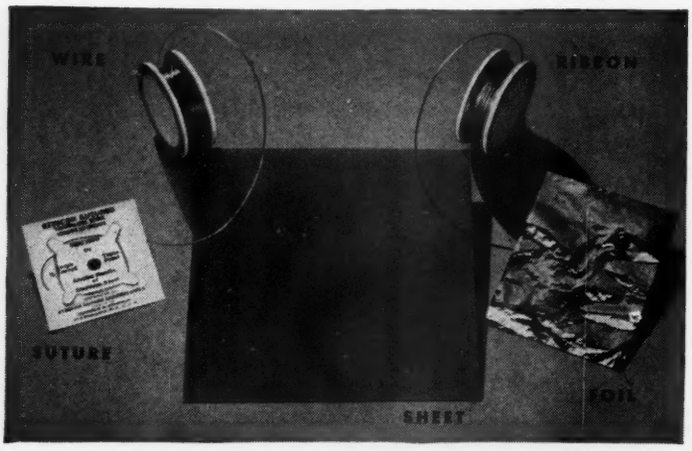

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In patients sustaining severe burns, the daily protein loss may be equivalent to 400 cc. of plasma.[2]

In a study embracing 23 burned patients, nitrogen balance determinations revealed excessive urinary nitrogen excretion. Nearly all patients were in negative nitrogen balance which was most marked during the first ten days.[3]

It thus appears that protein destruction and loss are prominent and potentially detrimental sequelae of trauma, and that every effort must be made to restore nitrogen equilibrium as quickly as possible to prevent the many deleterious consequences of protein depletion. The recommendation has been voiced that "whenever possible, protein losses or deficiencies should be corrected by oral feeding."[4]

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1 Howard, J. E.: Bull. Johns Hopkins Hosp., *74*:313 (May) 1944.

2 Co Tui, C.; Wright, A. M.; Mulholland, J. H.; Barcham, T., and Breed, E. S.: Ann. Surg. *119*:815-823 (June) 1944.

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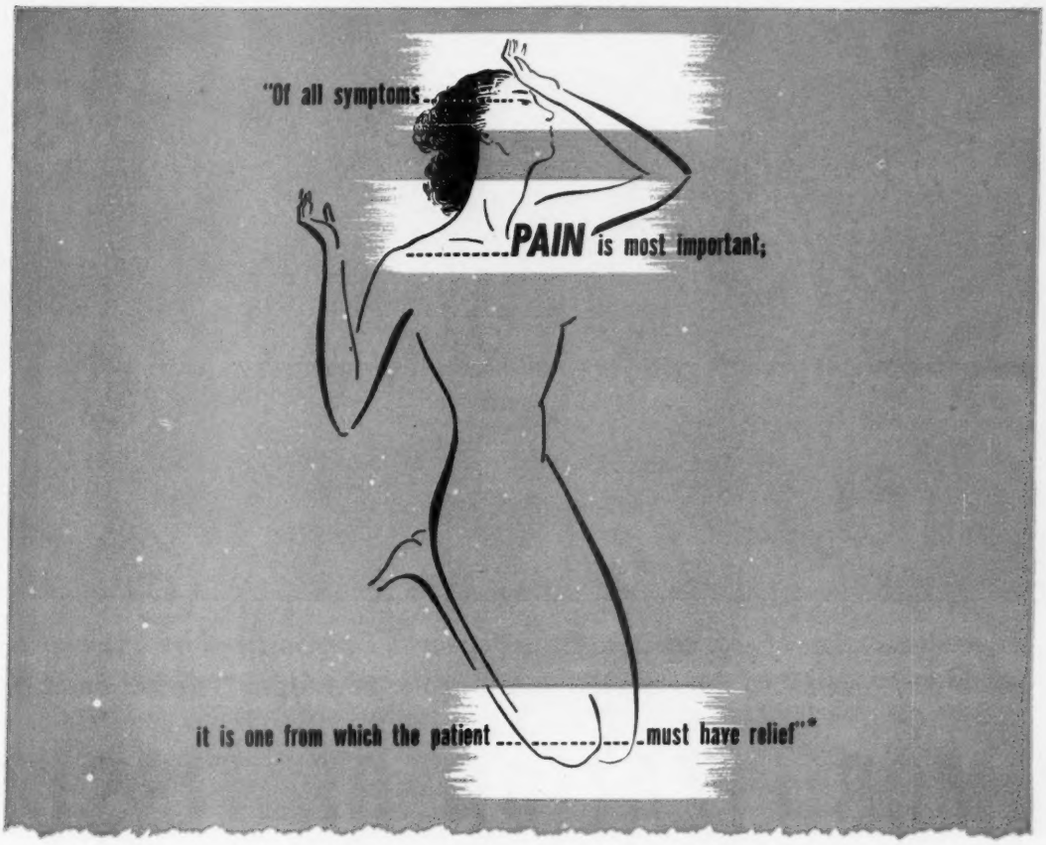

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*Handbook of Therapy, Chicago, American Medical Association, 1935, p. 58.

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VOL. LXXII DECEMBER, 1946 NUMBER SIX

Editorial

ACUTE SURGICAL CONDITIONS OF THE ABDOMEN

THE development of American surgery has been characterized by several great epochs, such as the era up to and including the time of Ephraim McDowell; the Watson, Long and Morton period, in which general anesthesia was introduced; the aseptic age of Lister and Pasteur; the period of the surgical giants, Halstead, Murphy, Senn and Ochsner; the clinic era of the Mayos and Crile; and, finally, the present epoch when improved pre- and postoperative care, chemotherapy, improved methods of anesthesia and early ambulation have proved a most important rôle in surgery.

In no field of surgery have the developments of the present era played a more important rôle than in that of acute abdominal surgery. The tremendous lowering of mortality in acute perforated appendicitis, which in the past thirty years has caused 500,000 deaths, is a triumph of chemotherapy. The sulfonamides and penicillin have largely eliminated the necessity for drainage in these patients and have consequently reduced to a fraction the incidence of complications such as obstruction, abscess formation, wound infection and eviseration. Chemotherapy has also reduced mortality, in acute perforated ulcer and in acute intestinal obstruction necessitating decompression or resection. By its use better results have been achieved in acute biliary tract surgery.

The introduction of ethylene, cyclopropane, pentothal sodium, curare, and, perhaps most important, improved methods of spinal anesthesia have greatly lessened the problems of the abdominal surgeon. Under spinal anesthesia the abdomen is perfectly relaxed whether one is closing an acute perforated ulcer, exploring the common duct or reducing a strangulated hernia.

Perhaps the most remarkable advancement that has come about in the past five years is early ambulation. It has been difficult to convince surgeons, as well as nurses, long accustomed to keeping their patients in bed for days and even weeks, that getting patients out of bed on the first or second postoperative day results in a far smoother and speedier convalescence. My only regret is that many years ago I did not disregard ideas which were accepted as facts regarding the advantages of prolonged bed rest in abdominal surgery. Early ambulation not only heals the incision speedily by improving the circulation but also reduces the necessity for catheterization and enemas. Normal peristalsis is induced, the patient is able to eat and enjoy food, gas pains are infrequent, duodenal suction and intravenous therapy are

required only in the more critical cases and pulmonary complications seldom occur.

Although great strides have been made in treating acute surgical lesions of the abdomen, the problem of diagnosis often remains a confusing and baffling one that can be solved only by an exploratory operation. For example, I know of no reported instance in which a correct preoperative diagnosis was made in a case of acute torsion of the omentum. Not many years ago I operated upon a boy for acute hemorrhage of a Meckel's diverticulum; this was the first recorded case in which a preoperative diagnosis (established by his family physician) was made of this condition. In 1927, I operated upon a man for acute obstruction and found several feet of thick hose-like jejunum. The diagnosis for this condition was not determined until 1932 when Crohn and his associates at Mount Sinai Hospital established acute regional enteritis as a distinct entity. The roentgenologist has so improved his technic that not infrequently he is able to detect Kantor's string sign in the terminal ileum, as well as other lesions of the small intestine, to aid the surgeon in locating the source of the trouble. Yet occasionally a gallstone may perforate and cause symptoms of acute obstruction far down in the small intestine. Or a young man may be admitted to the hospital in shock after a motorcycle accident; physical examination reveals nothing. Following the administration of blood plasma and further shock therapy he rapidly improves. Ten days later he returns to work, collapses on the third day and is rushed back to the operating room; a laceration of the liver that nature had temporarily walled off is revealed upon abdominal exploration.

The problems of the acute traumatic abdomen in this machine age are such as to bring gray hair to any surgeon, but we have made it a rule at our clinic to operate when in doubt.

Probably the commonest and yet the most confusing diagnostic problem for the thousands of American surgeons remains that of the lowly appendix. The fact that in the past as many as 45,000 cases of perforation have occurred in a single year seems to indicate that a great number of errors occur on the part of someone, the patient, the relatives, the physician or the surgeon. Too many fail to realize that the patient may act and appear well, may have a normal temperature, a normal blood count, but slight rigidity and only moderate tenderness on deep palpation, and yet have a retrocecal gangrenous appendix.

Delay in diagnosis is also largely responsible for the high mortality rate that still persists in acute intestinal obstruction. Earlier surgical intervention in attacking these conditions is imperative if the end results are to be improved.

The art of correct diagnosis has truly been termed the highest achievement of the surgeon. In no other part of the body is his experience and judgment so accurately tested as in the field of acute abdominal surgery. He must not only keep in mind the more important and frequent conditions discussed in this editorial, but must also be prepared to exclude acute lesions of the genitourinary and gynecologic systems. Nor should he forget that coronary disease may occasionally resemble gallstone colic and vice versa and, still more puzzling, that they may both occur in the same individual. He must also remember that pneumonia in children may simulate appendicitis and that enteritis may occur alone or with appendicitis. He should make frequent examinations of the urine to exclude pyelitis if the first specimen is negative.

Surgery in acute conditions of the abdomen is an intriguing and fascinating subject that constantly challenges the skill and ingenuity of the surgeon with its many complex problems.

Arnold S. Jackson M.D.

Original Articles

DIFFERENTIAL DIAGNOSIS IN ACUTE SURGICAL CONDITIONS OF THE ABDOMEN

HERBERT WILLY MEYER, M.D.
NEW YORK, NEW YORK

PHYSICIANS and surgeons are constantly confronted with the major problem of making a differential diagnosis in a patient suspected of suffering from an acute surgical abdomen. The surgeon is usually called in consultation by the physician who recognizes a surgical emergency. Frequently the decision must be made late at night when the physician sees the patient again or when the patient is admitted to the hospital.

The surgeon should be keen and alert to all possibilities and must make a serious and thorough attempt at diagnosis. There is usually an urgent need for surgical intervention.

A snap diagnosis may be magnificent and may make an impression on some, but it is unsafe and unsound. However, no one can recognize all the possible causes of an acute condition of the abdomen either before operation or before a postmortem examination unless he have the confidence born of youthful inexperience.

Early diagnosis is essential. If a patient has severe abdominal pains lasting longer than six hours and has previously been well, one must suspect a condition of surgical importance.

A history that the patient recently has received a hypodermic of morphine or some other anodyne must immediately make the surgeon suspicious of serious disease.

It is important to keep certain groups of diseases uppermost in one's mind when making a diagnosis, realizing however, that certain rarer conditions might be present. These groups are: (1) inflammations, (2) perforations, (3) colics, (4) hemorrhages, and (5) obstructions.

The examining surgeon must form the habit of a definite systematic routine examination in every acute abdominal case.

There are certain dicta which are well to keep in mind. Some of them are: (1) It is more important to put the examining finger in the rectum than a clinical thermometer in the mouth. (2) Palpating the pelvic peritoneum is more important than palpating the pulse. (3) Examination of the urine is as important as to inquire about the state of the bowels. (4) Palpation of the abdomen may present muscular rigidity but auscultation of the chest with a stethescope may show pleurisy or pneumonia.

The knowledge and application of the surgeon's training in anatomy and physiology are of the greatest importance in making a differential diagnosis. He must train himself to think anatomically and physiologically, just as the neurologists and neurosurgeons.

The voluntary muscles and the spinal nerves are the least variable factors in the abdomen.

Early symptoms in all groups may be the same since they all depend on the same cause, such as the shock to the solar plexus, as evidenced clinically by pain, vomiting and shock.

Pain is caused when irritated muscles are moved. Therefore, it is important to include the examination of the state of the

psoas muscle in a suspected case of acute appendicitis.

The anatomical distribution and course of the segmental spinal nerves is an important aid. One remembers that the testicles develop in the same region as the kidney, which explains that patients may have pain in the lumbar region and at the same time also have pain in the testicle. Dorsal distribution of the referred pain is important.

Similarly the diaphragm develops in the region of the cervical segments and later descends, dragging the phrenic nerve with it, which mainly comes from the fourth cervical root. This easily explains why lesions in the lower chest and upper abdomen present pain in the distribution area of the fourth cervical nerve, with referred pain over the shoulder, scapula and subclavicular area.

Conversely the pelvic nerves are not represented in the abdominal musculature and therefore patients with a pelvic peritonitis will not necessarily show muscular rigidity.

In the obstruction group of lesions the knowledge of physiology is more important than anatomy. Pain is caused by distention and stretching of the tubes by gas or fluid or by both. Severe colicky pains are caused by an obstruction or reflex spasm of a tube with local distention proximal to it. When strong peristaltic contractions take place, pain is caused. It is then referred to the sympathetic nerve center from which the specific nerve comes. It is also referred to the corresponding segmental spinal cord nerve and its distribution.

Thus colic of the small intestine causes pain in the epigastric and umbilical region. Colic of the large intestine causes pain in the hypogastrium. Biliary colic is felt in the right hypochondrium and the right subscapular region. Renal colic is felt in the lumbar region and radiates to the thigh and to the testicle.

Shock is a stage of an abdominal emergency, caused by the sudden stimulation of many nerve centers, as in a perforated ulcer or the stimulation of a few nerve centers as in biliary colic. Following this initial shock there may be a stage of reaction with a free interval, and this is followed by a stage which is peculiar to each specific lesion.

It is difficult at times to convince a patient and the family of the seriousness of the acute emergency because the first stage of shock may be a transient one. The ensuing free interval is but the calm before the storm, with a renewal of more severe and serious symptoms. Persistence of shock is important, and the pulse pressure may be evidence of the severity of the shock and the impending state of collapse.

METHOD OF MAKING A DIAGNOSIS

Every surgeon develops his own routine in performing an examination of a patient. It may be well to summarize a few points essential to this examination, and then to take up each point separately in greater detail. The essential points are:

1. History of the present illness
   - Age of patient
   - Time and mode of onset of attack
   - Pain and its first situation, shift, radiation, character and pain on micturition
   - Nausea and vomiting, whether before, same time or after onset of attack
   - Movement of bowels
   - Menstrual history
2. Past History
   - Whether patient had a similar previous attack
   - History of indigestion with relation to meals, jaundice, etc.
   - Confinements
3. Physical and clinical examination
   - Pulse, temperature, respiration, tongue and blood pressure
   - General appearance and attitude of patient
   - Abdominal examination
   - Rectal and vaginal examination
   - Chest examination
   - Reflexes—K-J, pupillary
   - Urine examination for albumin, sugar, pus and blood
   - *X*-ray film of abdomen and lower chest, if available and feasible

AGE

Age may be of help in thinking of possible diagnosis in the various differential diseases.

Disease	Age Factors
Acute intussusception	Usually under two years, may be at any age
Acute appendicitis	Most common in young adolescents, may occur at any age
Acute cholecystitis	Rare in childhood, more common in adult life
Acute pancreatitis	Seldom or never under middle age
Acute regional ileitis	More common in adult life
Perforated gastric or duodenal ulcer	Rare under fifteen years of age
Obstruction of large intestine	Rare before thirty, infrequent before forty, commonest over forty
Obstruction, strangulation of small intestine, especially through hernial apertures	At any time of life
Twisted pedicle of ovarian cyst	Adult life
Derangements of developing ovum and its environment	Child bearing period
Black widow spider bites	At any age, depending on locality of patient

PAIN

The site of onset of the pain, the shifting or the localization of the pain, its character and the radiation are all important factors in the various differential diagnoses.

It is well to remember that the pain is felt at the site of the inflammation if the parietal peritoneum is involved.

The presence or absence of localized pain can be either a good or bad sign. It is a good sign in acute appendicitis if there is localized pain as it means that the parietal peritoneum is involved and adhesions are more apt to develop. If there is no localized pain it may mean that the appendix is buried in the coils of the intestines, adhesions do not form as readily and spreading peritonitis may ensue. Of course this refers only to the first attack of appendicitis.

Diagnosis	Pain
Acute appendicitis	Arises in epigastrium and shifts to right lower quadrant. May awaken patient out of sleep, is gradual in onset increasing to a maximum, and is aching in character. If accompanied by painful micturition, appendix is likely to be a pelvic one.
Biliary colic	Arises in right hypochondrium. Radiates to the back and to tip of scapula, or top of right shoulder.
Renal colic	Starts in lumbar region. Radiates to corresponding thigh and testicle. Sharp, constricting and severe in character.
Intestinal colic	Starts in epigastrium. Intermittent, griping pain. Relieved by pressure of examining hand. Becomes general.
Gastric or duodenal ulcer perforation	Feeling of faintness at onset of pain in upper abdomen. May awaken out of sleep. Is excruciating in character at onset. Pain felt in location where flooding of peritoneal cavity began, then shifts downward as escaping contents pass downward and pain then becomes general.

Diagnosis	Pain
Acute pancreatitis	Feeling of faintness at onset. Agonizing in character in upper abdomen and then becomes more general.
Abdominal injury with rupture of intestine	Starts in epigastrium and shifts to hypogastrium as leakage descends downward.
Strangulation of intestine	Severe at onset and gradually becomes worse.
Torsion cyst pedicle	
Acute mesenteric thrombosis	
Ruptured ectopic gestation	Feeling of faintness at onset. Pain felt in lower abdomen.
Obstruction of colon	Pain starts in hypogastrium. Shifts to local site if mesocecum or mesocolon is short. Gradual culminating in crisis.

It is essential to note the influence of respiration on the pain. If there is pain during micturition, one must consider a pelvic abscess or inflamed appendix but the various kidney lesions such as pyelitis, kidney or ureter stone or acute hydronephrosis or pyonephrosis must be kept in mind. The pain in hydronephrosis or pyonephrosis is much duller in character than in the intraabdominal lesions.

VOMITING

Vomiting, like pain, is a reflex phenomenon. It is, therefore, of questionable value in differential diagnosis. It is caused by irritation of the nerves of the mesentery or of the peritoneum. It may be caused by tension in an involuntary muscle tube. Finally, it is caused by the toxemia of septic peritonitis.

Vomiting usually subsides within twenty-four hours after onset of an acute condition. If it then recurs, it is a serious sign and usually means the beginning of spreading peritonitis. If vomiting persists from the onset of the attack it is also a serious sign and usually means intestinal obstruction.

The relationship of the onset of pain and vomiting is of some diagnostic value.

Severe pain of biliary or renal colic	Vomiting starts early
Intestinal obstruction	Long interval between onset of pain and onset of vomiting.
Obstruction upper intestine	Shorter interval between pain and onset of vomiting.
Obstruction of low intestine	Long interval between pain and onset of vomiting.
Appendicitis	Pain is usually present three to four hours. possibly twenty-four hours before onset of vomiting.

The frequency of vomiting is of significance. Frequent vomiting in acute appendicitis often means early perforation if it continues early in an attack. If vomiting is frequent later in the attack, it means spreading peritonitis. In the case of a ruptured gestation and in perforated ulcer there may be no vomiting at all.

The character of the vomiting is likewise significant.

Acute gastritis	Stomach contents and little bile
Colics	Usually contains bile
Torsion or stangulation with sympathetic shock	Retching with little vomitus
Intestinal obstruction	Stomach contents, then bile, then greenish-yellow and finally fecal with fecal odor. This always means obstruction, either paralytic or mechanical.

Some patients rarely vomit but may have nausea with loss of appetite. Pain in the abdomen with loss of appetite in a child previously well should always make a sur-

geon very suspicious of acute appendicitis and it must be ruled out.

BOWELS

One cannot put much reliance on constipation or diarrhea. One should always ask the patient when they were conscious of last having passed flatus spontaneously. The question of whether the patient took or was given a cathartic by mouth, or whether an enema was given is of great importance. If there is onset of constipation, pain and flatulence, the patient must be carefully observed. Tenesmus frequently is the symptom of a pelvic acute appendicitis, perhaps with abscess, or of a simple pelvic abscess. Blood and mucus in the stool with pain is suspicious of a possible intussusception.

MENSTRUATION

Always ask a patient the date of onset of the last menstrual period and its character, whether different than usual or whether a change in regularity. If suspicious, ask the time of last intercourse and whether there is a vaginal discharge present. Consider threatened abortion, ectopic gestation if the pain occurred with the period, and especially if this pain is unusual or uncommon for the patient.

PAST HISTORY

Always ask whether the patient has ever had a previous similar attack. Inquire about indigestion and its relation to meals. Question the patient about loss of weight, previous jaundice, blood in the stools or in the urine, or vomiting of blood. Previous illnesses must also be inquired into such as typhoid fever, operations for appendicitis, with or without peritonitis and drainage, pneumonia, etc.

PHYSICAL AND CLINICAL EXAMINATION

Pulse. The pulse may be misleading in the differential diagnosis of an acute surgical condition of the abdomen. After an abdominal injury the pulse should be taken and recorded hourly. It rises rapidly in hemorrhage. It may be high at the onset after perforation and then go lower, only to rise again when peritonitis sets in. In the early stages of even a severe acute condition the pulse may be normal.

Temperature. This also may be misleading in an acute condition. In inflammations the temperature may not be elevated in the early stages. High temperature with chills and presence of herpes on the lips usually means chest disorders.

Early subnormal temperature	Acute pancreatitis, high later. Aute strangulation, perforated ulcer, hemorrhage.
Early normal temperature then slowly rising to 100°–101°F.	Acute appendicitis
Sudden rise in temperature and drop to normal or subnormal	Perforated appendicitis due to absorption of toxins.
Early temperature 104°–105°F.	Thoracic or kidney disease, rarely in appendicitis.

Respiration. The ratio of the pulse to respiration may be of some value in differential diagnosis. Also whether the rate of respiration is out of all proportion to the abdominal symptoms, and whether the accessory muscles of respiration are active, as the ala nasi. The respirations may be 40 to 50 and the pulse only 100 as in early thoracic lesions.

Intrathoracic lesion	Ratio pulse to respiration less than three to one
Intra-abdominal lesion	Ratio pulse to respiration more than three to one

It may be a fair criterion to say that if the respiration is twice the normal at the onset of the attack, it is probably a thoracic lesion. It is always well to keep in mind that pressure against the diaphragm by an acutely dilated stomach, peritonitis, hemorrhage, or by obstruction with distention, will cause the respiration to be rapid, shallow and labored.

Tongue. It is always a good habit to ask to see the condition of the tongue. It is valuable to note whether it is dry or moist, both at the lateral borders of the tongue and in the center. A dry tongue means dehydration due to loss of fluids, or to sepsis and toxemia.

Acute appendicitis and obstruction	Tongue is coated and there is usually a foul breath

General Appearance. A great deal can be learned in the differential diagnosis of an acutely ill patient by the facial expression and the position the patient takes in bed when the examining surgeon enters the room.

Perforated ulcer Acute pancreatitis Acute strangulation of intestine	Patient is pale with cold perspiration and lies very quiet
Abdominal injury with hemorrhage Ruptured ectopic gestation	Deathly pallor and air hunger with restlessness of patient
Severe toxemia of peritonitis	Sunken cheeks, eyes, and dull gaze. Patient lies immobile, possibly with flexion of knees and thighs.
Colics	Anxious expression, evident extremely severe pain and marked restlessness, usually rolling around in bed.

Examination of the Abdomen. Personally I have always been in the habit of asking the patient to point with one finger to the point of maximum pain. Following this a rapid inspection of the abdomen is done. Look for general or local distention and for evident distended loops or coils of intestines. Also whether there is any evidence of visible peristalsis if distended coils are visible.

Distention of upper abdomen only	Acute dilatation of the stomach
Distention of the midportion of the abdomen with empty lumbar gutters	Small intestinal obstruction or paralytic ileus
Distention midabdomen and lumbar gutters	Large intestinal obstruction or general paralytic ileus

Inspect all the hernial apertures for a swelling and never overlook the femoral ring. Think also of the possibility of internal hernias, or a volvulus, causing the obstruction. Look for evidence of abdominal tumors and swellings.

Limitation of abdominal respiration found in	Acute perforations, biliary colic, pancreatitis and appendicitis.

Palpation of the abdomen must always be most gentle with the warmed hand placed lightly upon the abdomen, using the finger tips gently to elicit evidence of voluntary or involuntary muscle spasm and rigidity. It is well to start the examination at a point distant from the point of maximum pain. Initial examination may be done with the knees extended but if spasm is found it should be repeated with flexion of the knees. Rigidity must be determined to be either voluntary or involuntary, localized or general. Muscular rigidity is a visceromotor reflex. It is due to stimuli received by the spinal cord from a diseased organ. It is nature's effort to protect the underlying diseased organ. It will depend a great deal on the chemical consistency of the fluid which is poured out into the peritoneal cavity.

Gastric contents	Marked rigidity
Intestinal and bile content	Moderate rigidity
Urine	Less rigidity
Blood	No rigidity. Later when blood clots it becomes more irritating.

Differentiation between Voluntary and Involuntary Rigidity. True involuntary rigidity never relaxes however gentle the

examination, however one distracts the patient's attention from the examination, nor is there any relaxation during any part of the respiratory effort. It is an absolute sign of serious trouble, usually with some form of chemical irritation of the peritoneum. It is almost always a strong indication for surgery. Conversely its absence is of no importance as there may be gross hemorrhage within the abdomen without any evidence of abdominal rigidity. The voluntary type of rigidity almost always shows a momentary relaxation of the muscular wall during the respiratory effort. In inflammations the involuntary rigidity becomes more marked as the parietal peritoneum becomes more involved.

Tenderness. Local deep tenderness is always a very valuable sign. It is not always due to pressure directly on the inflamed organ. It is either due to hyperesthesia, which is superficial, and a true viserosensory reflex, or due to irritation of the local peritoneum from stimulation of the subserous nerve plexuses connected with the cerebrospinal system.

Superficial abdominal wall tenderness	More common in respiratory lesions
Local tenderness	Appendicitis Cholecystitis Pancreatitis Salpingitis
General tenderness	Peritonitis

Palpation and bimanual palpation of the lumbar regions detects renal lesions and other swellings. Turning the patient, if possible, to the opposite side and palpating the lumbar regions and along the iliac crest in the iliac flare is of value. At this time one can test for psoas spasm by trying to bring the extended leg backward. If there is tenderness and rigidity in the flank one must consider retrocecal or retrocolic appendix, perinephritic abscess, inflamed kidney, lumbar abscess and hydro or pyonephrosis.

Hyperesthesia. This is a viscerosensory reflex. Superficial skin hyperesthesia elicited by either pinching the skin lightly or by a light pin stroke over the skin is not too reliable a test. In certain cases, however, it is difficult to determine the organ at fault and then the test may be of diagnostic value. A clinician may then be enabled to make a keen diagnosis. If positive, it may be of value; if negative, it means nothing. Points of increased skin sensitivity are as follows:

Gall bladder	Mammary line below costal margin
Appendix	Mammary line below level umbilicus
Fallopian tubes	Mammary line above inguinal ligament
Stomach and duodenum	Epigastrium from xiphoid to umbilicus
Small intestine	Below umbilicus
Large intestine	Mid-line suprapubically

Liver Dullness. Examination for presence or absence of liver dullness is of importance. As a positive sign it is important. As a negative sign it is of no significance. If positive, it means a rupture of a hollow viscus. There will be resonance from the fifth rib to the costal margin in the mammary line and from the seventh to the eleventh ribs in the midaxillary line.

Free Fluid. Patients are usually too ill to be examined routinely for free fluid in the abdomen. Shifting dullness may be found in large intraperitoneal hemorrhages as in ruptured ectopic gestation. It may, of course, occur in a rupture of a hollow organ containing much fluid as a full stomach, full bladder or the rupture of a large ovarian or hydatid cyst.

Abdominal or Pelvic Masses and Swellings. These are often noted especially in inflammatory lesions. An appendix mass or gallbladder can sometimes be easily palpated. The danger of the rupture of an appendix abscess by too firm palpation through overlying rigidity must always be avoided. A mass can be felt over an intussusception, malignant growth with obstruction, fecal masses, and rarely over a

large gallstone in the intestines that has ulcerated through the wall of the gallbladder into the intestine. Omental masses, the policemen of the abdomen, can also be palpated.

Pelvic Examination. The suprapubic region must be palpated and percussed. In the differential diagnosis an overdistended bladder must be kept in mind. If in doubt always pass a catheter. An enlarged uterus also comes into consideration, as well as an ovarian cyst or a pelvic abscess. A bimanual vaginal examination is necessary in every non-virginal female.

Rectal Digital Examination. This should never be forgotten. Pain on pressure with the examining finger tip and pain on movement of the finger tip is important. It must not be pain due to the discomfort of the examination. The examining finger should be introduced under direct vision with good light, slowly, gently and well lubricated. Blood, mucus or pus on the withdrawal of the gloved finger must be noted. If present, suspect intussusception, perforation of a pelvic abscess into the rectum or tumor. Rectal examinations are especially of value in pelvic inflammatory lesions. A mass in the anterior pelvis may be a pelvic abscess, a lateral mass is more apt to be an appendiceal abscess. A high rectal examination may detect a tumor and a stricture.

Auscultation of the Abdomen. This is also of value in the differential diagnosis.

Silent abdomen	Paralysis of the intestines due to ileus or peritonitis
Gurgling abdomen with pain	Intestinal obstruction

General Physical and Clinical Pathological Examination.

Chest examination	This is essential in every case and requires careful examinations of lungs heart and pericardium.
Reflexes	Patella and pupillary reflexes must be tested to rule out tabetic gastric crises.
Urine examination	Blood, pus, albumin, sugar and bacteria are important to look for. The patient should be catheterized if necessary before surgical intervention is resorted to.
Blood count	Always advisable but do not put too much reliance on it. If elevated it helps to confirm the diagnosis. If normal it does not mean anything in the presence of other signs of an acute surgical condition. It should never be taken as a criterion for or against surgical intervention.
Blood pressure	The pulse pressure is indicative of the severity of shock and the state of impending collapse.

X-ray Examination. If the patient is in good enough physical condition and *x*-ray examination is easily available, a film of the abdomen and chest may be of real help in making a differential diagnosis in difficult cases.

Intestinal obstruction	
Small intestines	Distended stepladder coils of small intestine
Large intestines	Distended and collapsed large bowel. It may show the site of obstruction
Intussusception	A barium clysma will confirm the suspected diagnosis
Perforation of hollow organ	*X*-ray may show evidence of a pneumoperitoneum

GROUPS OF CASES

Having layed the foundation for the differential diagnosis in an acute surgical condition of the abdomen, it will be wise to say something about the various groups of diseases and to discuss in greater detail the differential diagnosis. More attention will be paid to acute appendicitis and its differential diagnosis as it is by far the most common cause. Abdominal injuries should also be taken up in greater detail.

Inflammations. In this group the onset is not as sudden. The patient usually complains of not having felt as well for a day or so before the onset of the acute symptoms. The pain gradually increases to a maximum over a period of hours. It is not as severe at the onset as it is in the colics, perforations and some forms of obstruction. The temperature rises slightly and then may go back to normal. One almost always finds local tenderness, be it by abdominal palpation or by vaginal or rectal examination. Tenderness is one of the most important signs. The muscular rigidity is of the involuntary type and increases in degree and in extent as the peritoneum becomes more involved. A local swelling may become palpable, such as an inflamed gallbladder, appendix or Fallopian tube, or omentum wrapped around and adherent to one of these inflamed organs.

One of the main and more important differential diagnoses is between acute appendicitis and acute cholecystitis. The question of an empyema of the gallbladder comes into consideration. Some of the more valuable differential points are:

	Acute Appendicitis	Acute Cholecystitis
History	More sudden onset	Gradual onset with history of indigestion
Local rigidity and tenderness	Over McBurney's point. Depends on whether organ is situated low or high	Over hypogastrium right side and below costal margin. Depends whether gallbladder is situated high or low
Cutaneous hyperalgesia	Mammary line just below level of umbilicus	Mammary line just below costal margin
Respiration	An appendix abscess stays stationary	Gallbladder mass moves downward on respiration
Shape of swelling	Rounded edge above	Rounded edge below
Tympany on percussion	Tympany between appendix swelling and liver	No tympany between gallbladder swelling and liver

In the differential diagnosis between acute appendicitis and acute salpingitis the following points are important:

	Acute Appendicitis	Acute Salpingitis
History	More orderly sequence of events with pain around umbilicus	Less orderly onset with initial pain below level of umbilicus. History of pelvic troubles
Local rigidity and tenderness	Over McBurney's point in an iliac appendix	No rigidity but tenderness on pelvic vaginal examination
Cutaneous hyperalgesia	Mammary line just below level of umbilicus	Mammary line just above inguinal ligament
Abdominal respiration	Some limitation	No limitation
Vaginal examination	No discharge	Usually some vaginal and cervical discharge

Congenitally the inflamed appendix may be situated in different portions of the abdominal cavity. Much depends on whether complete rotation and normal descent of the cecum has taken place. This fact will also determine whether the appendix lies free in the peritoneal cavity or whether it

is partially or wholly covered by perietal peritoneum and lies completely retroperitoneal. Depending then upon the position of the appendix various pathological conditions have to be considered in the differential diagnosis of acute appendicitis. They are:

Ascending Appendix	Iliac Appendix	Pelvic Appendix
Duodenal ulcer	Leaking perforated ulcer	Meckel's diverticulitis
Cholecystitis		Diverticulitis with abscess
Perforated gallbladder	Ileocecal tuberculosis	Perforated typhoid ulcer
	Cecal carcinoma	Ureter calculus
	Mesenteric lymphadenitis	Intestinal obstruction
	Psoas abscess	Female patients
	Hip disease	Ectopic gestation
		Salpingitis
		Ruptured pyosalpinx
		Twisted pedicle of
		Hydrosalpinx
		Ovarian cyst
		Pedunculated fibroid

Intussusception. This usually occurs in young children. It may occur also in adults and then usually is caused by a neoplasm, benign or malignant, within the lumen of of the gut This invaginates itself into the bowel and draws along the intestine in an intussusception. There is quite some pain at the onset, accompanied by initial reflex vomiting and a mass can frequently be felt along the colon at the site of the intussusception. Blood and mucus are present with some pain on bowel movement. It may also be found on the examining finger on rectal examination. A barium clysma, if available and feasible, will verify the diagnosis.

Acute Pancreatitis. This acute surgical emergency is one of the rare ones to keep in mind. Still one must always be cognizant of its possible presence. It is sudden in onset, severe in character, and a true surgical emergency. The pain in the epigastrium is severe and is often mistaken for a perforation. There is early reflex vomiting, shock, subnormal temperature at first followed later by high temperature and a very rapid pulse. Tenderness and local rigidity are confined at first to the epigastrium over the pancreas and later become more general. Abdominal respiration is limited in extent. The blood count is high. The disease accounts for less than 1 per cent of all acute abdominal conditions.

Regional Ileitis. This condition is rarely an acute surgical emergency, although it is possible that a perforation of the ileum without sufficient adhesions could present the picture of a spreading peritonitis. The condition is present for a longer time and when it becomes acute it resembles acute appendicitis. There is right lower quadrant pain and the patients have fever, vomiting, and sometimes diarrhea and a secondary anemia. Internal perforations occur but adhesions are usually present. Later in the disease external perforations may occur.

Hemorrhages. If the hemorrhage occurs into the lumen of the alimentary canal or into any organ lined by mucous membrane, the blood will appear at one of the orifices. If the bleeding is into the stomach, it will appear at the mouth; if it is from the upper alimentary tract or from the bowels, it will appear at the rectum. Uterine bleeding will appear at the vagina. Hemorrhage into the peritoneal cavity is more serious as it is not irritating to the peritoneum in the early stages. It may, therefore, continue unnoticed. The abdomen is soft and not distended. The patient, however, becomes restless and the pulse rises rapidly. If there has been a sudden onset of these symptoms with pain in the abdomen, think of hemorrhage. The patient becomes increasingly pale. Repeated blood counts, especially the hematocrit deter-

mination, is of real value. It will help to determine the extent, degree and persistence or cessation of the bleeding. Auscultation of the abdomen is of value as it will usually be a silent abdomen. The most common causes are trauma and ruptured ectopic gestation.

The main differential diagnostic points are

Ruptured ectopic gestation	Restless, no abdominal rigidity
Perforated ulcer	Patient lies absolutely still with marked abdominal rigidity

Perforations. The history in the majority of cases will be of value. Indigestion has usually been present although this history may be entirely lacking. The onset is sudden, may occur during sleep and awaken the patient. It may also occur after bodily strain and exertion, or without any warning or predisposing cause. The pain is excruciating and there is initial shock. This shock may soon pass off. Involuntary abdominal muscle rigidity is present over the entire abdomen. The pain at the onset is usually at the site of the organ that perforates, and then becomes general as leakage and descent of the same takes place. Peritonitis sets in. Free gas in the abdominal cavity is frequently present. It can be found while examining for liver dullness by percussion. It may be seen as pneumoperitoneum by *x*-ray examination. It accumulates usually under one or both leaves of the diaphragm, especially in the erect posture. A fluid level may be present. Limitation of abdominal respiration is marked. Testing for cutaneous hyperalgesia may be of value in a few cases in determining the organ at fault. It may help in difficult diagnostic cases to make a correct preoperative diagnosis. Operation and early operation alone will definitely show the organ at fault. The possibility of perforation in a retroperitoneal portion of one of the hollow organs must be kept in mind. It is important to make the differential diagnosis between perforation of a gastric and duodenal ulcer, biliary and renal colic and conditions as gastric crisis of tabes dorsalis and thoracic disease as pleuropneumonia. In the former, surgery is indicated, while in the latter (tabes and pneumonia) surgery is distinctly contraindicated.

Colics. These are usually sudden in onset, sudden in cessation and equally sudden in recurrence. Reflex vomiting occurs. The pain is at first felt near the structure at fault. Radiation of the pain is of great diagnostic value.

Biliary colic	Pain referred upward into the back, tip of the scapula, top of the shoulder. There is limitation of abdominal respiration.
Renal colic	The pain in the lumbar region is referred downward to the thigh and testicle of the corresponding side. There is no limitation of abdominal respiration. Frequency of urination occurs and there are red blood corpuscles in the urine.
Intestinal colic	The pain occurs all over the abdomen. Pressure of the examining hand on the abdomen sometimes relieves the pain, which is not the case when peritonitis is present

The hollow tubes composed of smooth muscle fibers which cause these colics if obstructed are: stomach, intestines, cystic, hepatic and common bile duct, ureters, uterus and Fallopian tubes and pancreatic duct.

Obstructions. There are very distinctive signs and symptoms in intestinal obstruction. The diagnosis should be made as early as possible as surgical intervention is usually indicated. The judicious use of the

Miller-Abbott tube is of value in deferring surgical intervention. It deflates the intestine and reduces the toxemia caused by absorption of the poisonous intestinal contents. Operation can be held in abeyance for a while, but if the Miller-Abbott tube does not pass readily its use should not be persisted in. Surgery is indicated.

Persistent vomiting from the onset of an attack with cessation of the passage of flatus and progressive abdominal distention is a signal for intervention, with a diagnosis of obstruction. If flatus does not pass together with dark intestinal content within twenty-four hours in spite of enemas, well given colonic irrigations and the careful use of prostigmin, the diagnosis of obstruction can be made. With the development of distention, individual loops of intestines may become visible if the abdominal wall is not too thick. Painful peristalsis may be seen. Auscultation of the abdomen is of value, as the characteristic gurgling and passage of fluid and gas through large distended coils of intestines may be heard. Fecal vomiting should never be mentioned as a symptom of obstruction. It is a terminal symptom, and the diagnosis should be suspected and should be made before the fecal vomiting sets in. The obstruction should have been relieved beforehand.

If obstruction is suspected in the differential diagnosis, the abdomen should be carefully examined for previous operative scars with possible adhesions and bands. A previous acute appendicitis with or without drainage should arouse suspicion.

Strangulated loops of intestines through the external hernial apertures, inguinal, femoral, umbilical, ventral, must always be looked for. At the same time strangulation due to internal volvulus and internal hernias must be considered.

X-ray examination with a film of the abdomen will show the typical stepladder distended loops of small intestine, and the distended or collapsed large intestine.

Certain conditions closely simulate the primary obstruction of the large bowel and come into consideration in the differential diagnosis. These are colitis with distention; paralytic ileus of the bowel, reflex ileus; uremia, and reflex paralytic ileus due to peritonitis.

EARLY DIFFERENTIAL DIAGNOSIS IN ABDOMINAL INJURIES

If there is a history of abdominal injury however slight it may have been, a serious intra-abdominal injury must always be considered. The patient should preferably be hospitalized and must be under constant careful observation. An hourly pulse record must be kept. Repeated blood counts and hematocrit determinations must be taken to give a clear picture of what may be occurring within the abdomen. The two common differential conditions are hemorrhage from solid organs and rupture or tears of hollow organs. If either of these conditions occur within the peritoneal cavity or in the retroperitoneal space, they are of equal importance. Injury to the liver, spleen, pancreas or the kidneys cause hemorrhage. If the kidneys are injured, there is also a leakage of urine. Injury to the hollow organs as stomach, intestines, gallbladder, ureters and bladder cause leakage of the contents with shock and peritonitis.

Following injury to the abdominal wall rupture of the intestine is the most common damage. As seen in war surgery, as well as civilian surgery, tears of the intestines may temporarily seal themselves off, while the bowel is reflexly paralyzed. After an interval of time or after taking nourishment peristalsis again sets in. The sealed off tear will begin to leak and peritonitis will follow.

The possibility of retroperitoneal rupture of the duodenum or of the colon with hemorrhage, must be kept in mind. Emphysema and cellutitis must be looked for at the time of operation. If found the condition must be corrected in order to save the patient.

If shock is still present three hours after an abdominal injury, the probability of some serious injury to some organ is likely. There will be pain, vomiting, local

or general rigidity, tenderness, advance in pulse rate, shallow respiration, restlessness if due to severe hemorrhage, obliteration of liver dullness and evidence of free fluid in the abdomen. Increase in temperature will follow.

A very important differential point to ask the patient is whether the injury occurred shortly after eating or whether it occurred before or just after emptying the bladder. A distended stomach or bladder is more apt to rupture than one that is empty.

If an injury to the bladder is suspected, a catheter should be passed. If no urine is obtained, a little saline may be gently injected. If materially less in quantity than what was injected is recovered from the catheter, a rupture of the bladder can be suspected.

In the differential diagnosis after injury the thorax must be carefully examined. Fractured ribs, pneumo or hemothorax, hemo or pneumopericardium must be looked for as these may give evidence of abdominal signs.

MEDICAL CONDITIONS IN THE DIFFERENTIAL DIAGNOSIS

There are finally various pure medical conditions that must be considered. Their presence may cloud the diagnosis. A surgical condition of the abdomen may be suspected when only a medical condition exists. The most common of these are:

Intrathoracic lesions
- Acute basal pneumonia,
- Acute diaphragmatic pleurisy,
- Acute pericarditis,
- Angina pectoris and
- Acute pneumopericardium.

Renal disease
- Uremia. Always remember its possibility.
  - Urine must be examined in every case.
  - Uremia may simulate large intestinal obstruction.

Spine and spinal cord lesions
- Tabes dorsalis with gastric, intestinal and renal crises. Pupillary and patella reflexes to be examined.

Blood diseases
- Leukemia. Will show enlarged spleen
- Acute infectious mononucleosis

General diseases
- Influenza
- Malaria
- Diabetes, impending coma
- Typhoid fever
- Tuberculous peritonitis
- Gonococcic peritonitis (abdominal puncture may aid in diagnosis)
- Ptomaine poisoning

Retroperitoneal conditions aside from acute pancreatitis
- Dissecting abdominal aneurysm
- Rupture of aneurysm of aorta or other abdominal vessel
- Retroperitoneal bleeding from kidney tumor or retroperitoneal tumor as sarcoma
- Retroperitoneal extravasation of bile or urine
- Pelvic infection which is subperitoneal due to extravasation following traumatic rupture of rectum at time of enema, etc.
- Retroperitoneal hemorrhage from fracture of contingent bones.

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DIFFERENTIAL DIAGNOSIS OF SURGICAL RENAL LESIONS*

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THE differential diagnosis of surgical renal lesions is here considered particularly from the standpoint of the general surgeon rather than the urologic specialist. Perforce, the abdominal surgeon too often only encounters the repetitious, vague abdominal pain frequently found on the right side and to which clinical picture the diagnosis of "chronic appendicitis" or "abdominal adhesions" is erroneously given. Yet appendectomy or exploratory laparotomy fails to relieve the existing symptoms and subsequent investigation commonly discloses unsuspected urologic disease. It is not uncommon to find some variety of ureteral obstruction or a renal lesion. (Fig. 1.)

FIG. 1. Stricture of the right ureter in a three-year old child whose abdominal pains had caused appendectomy to be done for "recurrent appendicitis." All symptoms were subsequently relieved by systoscopic dilatation of the ureter.

The diagnostic difficulties are at once apparent when it is recognized that practically every renal surgical disease may present a similar clinical picture although it is often not noted in the usual text book description. Thus chronic renal tuberculosis may simulate non-tuberculous chronic pyelonephritis, with secondary vesical involvement. The resulting urinary frequency causes the diagnosis of chronic cystitis to be made and the renal lesion overlooked. With the passage of clumps of tuberculous débris, the resulting renal colic may simulate ureteral stone; a partially or completely occluded tuberculous kidney with its advanced surrounding fibrolipomatous perinephritis and periodic hematuria, may closely simulate renal tumor.

In all cases, careful urinalysis of a properly collected specimen is of paramount diagnostic importance. When urologic disease and particularly when infection is suspected or must be considered in the differential diagnosis, examination of the casually voided specimen is essentially worthless. In over twenty-five years we have never examined a voided specimen from the female but insist on catheterization in both the newborn and the aged. Only by aseptic catheterization can bacterial and cellular introital contamination be avoided. In the male a satisfactory specimen can be obtained by washing the exposed glans penis and external meatus with an antiseptic solution such as oxycyanide or bichloride of mercury 1 :1000, after which the patient passes a few cubic centimeters of urine; into the continuing urinary stream

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Fig. 2. Characteristic roentgenographic findings in perinephric abscess. Noteworthy are the lateral sclerosis away from the side of the lesion and the obliteration of the shadow of the isolateral psoas muscle (left).

a sterile receptacle is introduced for specimen collection. This avoids extraneous cellular or bacterial contamination and if the procedure cannot be carried out satisfactorily, catheterize. In the subsequent examination of the urine we are interested primarily in the pus, blood and bacterial content, slightly less in the sugar content, and least of all in the albumin and cast elements. Yet the demonstration of pus casts at once establishes the presence of suppurative pyelonephritis. Cure of urosurgical disease implies a restoration of the urine to normal, and this includes sterilization as attested by at least two negative cultures of properly collected specimens. This dictum holds whether the basic disease is chronic pyelonephritis, infected hydronephrosis, renal tuberculosis, calculus or tumor. In addition to these conditions will be considered acute renal infection and congenital malformation.

SURGICAL NON-TUBERCULOUS RENAL INFECTIONS

As a rule the diagnosis in non-tuberculous renal infection is either acute or chronic "pyelitis." Properly pyelitis denotes inflammation of the kidney pelvis, yet to date such inflammation without involvement of the renal parenchyma has never been demonstrated. In renal infection, the usual lesion is an interstitial suppurative nephritis which may exist alone or as a complication of another condition such as stone or obstruction. In persistent acute renal infection, failure of intensive medical therapy (sulfonamide, penicillin, streptomycin) to bring about striking improvement in three to four days, calls for complete urologic examination by which the probable anatomic diagnosis will be made. This investigation will usually reveal infected hydronephrosis, generally

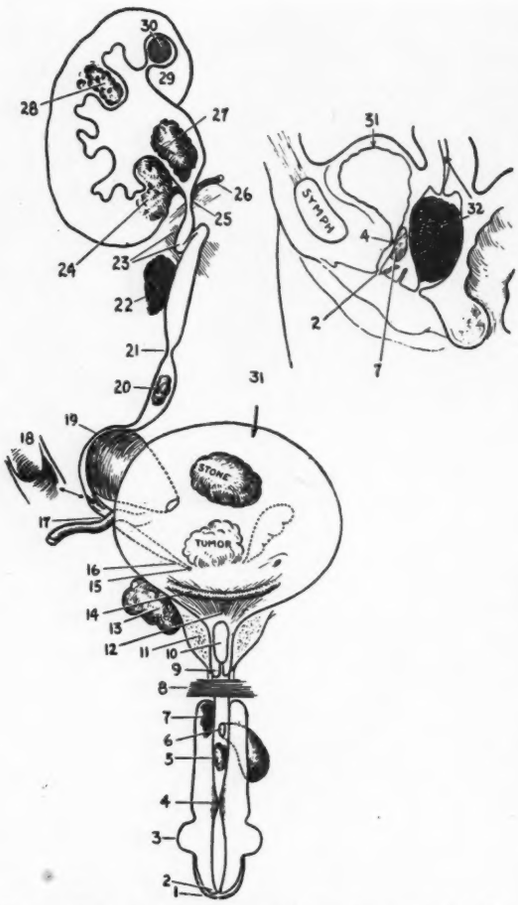


Fig. 3. Causes of urinary obstruction and, by the same token, hydronephrosis. 1, Phimosis; stenosis of prepuce; 2, stenosis of urethral meatus; 3, paraphimosis; 4, urethral stricture; 5, urethral stone; 6, urethral diverticulum; 7, periurethral abscess; 8, external sphincterospasm; 9, congenital valves of posterior urethra; 10, hyperthrophy of verumontanum; 11, prostatic anscess or growths; 12, contracted bladder neck; median bar; 13, periprostatic abscess; 14, mucosal fold at bladder outlet; trigonal curtain; 15, stricture of ureteral meatus; ureterocele; 16, ureterovesical junction stricture; 17, vascular obstruction of lower ureter; 18, congenital ureteral valves; 19, ureteral obstruction by vesical diverticulum compression; 20, ureteral stone; 21, ureteral stricture; 22, peritureteritis or tumor; 23, ureteral kink; periureteral fibrous bands; 24, renal tumor; 25, ureteropelvic junction stricture; 26, aberrant vessel obstruction of upper ureter; 27, pelvic stone; 28, renal tuberculosis (secondary obstructive lesions consequent thereto); 29, stricture of calyceal outlet; 30, calyceal stone; 31, neuromuscular vesical disease; 32, urethral compression by hematocoplometra.

consequent to blockage of the pelvic outlet or upper ureter by stone, stricture, malposition, an aberrant vessel, or perirenal abscess. Also there may be either massive metastatic focal infection (pyemic kidney), or solitary renal abscess (renal carbuncle). Sometimes the obstruction can be relieved and the organ preserved but here the surgeon replaces the kidney with the reservation that prompt secondary nephrectomy may be demanded or late secondary renal sclerosis may be the cause of hypertension in later months. Sometimes surgical exposure discloses massive renal suppuration and, when unilateral, nephrectomy rather than simple renal drainage is required. Occasionally, drainage of a perirenal abscess or coring out and drainage of a solitary renal abscess (renal carbuncle) is curative.

Any retroperitoneal edema-producing process such as perinephric abscess will roentgenographically obscure the isolateral psoas muscle outline. (Fig. 2.) As a rule, in perirenal abscess compensatory muscle spasm of the isolateral erector spinae group produces lumbar curvation away from the site of the lesion. When a patient presents (1) the picture of acute intra-abdominal disease but with unilateral renal tenderness and spasm of the overlying loin muscles, and (2) has a history of cutaneous infection (boils, carbuncles, furuncles and paronychiae) during the past three months, the roentgenologic demonstration of obliteration of the psoas muscle outline together with spinal curvature toward the opposite side, at once suggests perinephric abscess. (Fig. 2.) Yet retrocecal appendicitis with edema may produce essentially the same roentgenogram and the diagnosis appendicitis rendered more likely by the urographic demonstration of a normal upper urinary tract.

Moreover, the same picture of acute surgical disease is presented by the rare renal thrombosis or infarction, but this occurs more often in the young, notably infants. This condition is generally immediately preceded by acute ileocolitis and the renal onset is acute with abdominal pain, rapid renal enlargement, tenderness, variable shock and prostration. Here the pyelo-

Fig. 4. Hydronephrosis due to ureteral obstruction by carcinoma of the broad ligament. Here the parenchyma has been reduced to a thin shell. Longitudinal ureteral dilation has resulted in several secondary kinks in the upper end of the duct. The clinical manifestations were pain and mass in the side with scatter leucocytes in the urine.

graphic outline may be obliterated by the intense massive parenchymal swelling and at operation (when the precise diagnosis will most often be made) the kidney is found converted into a large blood clot-like mass. Nephrectomy is the treatment and to be life-saving must be immediate. I have successfully performed nephrectomy for renal thrombosis in two boys twelve and thirty-three days of age.

In short, when hyperacute renal infection persists, and especially unilaterally, prompt urologic examination is indicated and the resulting demonstration of infection, pyelographic distortion or obliteration will suggest the probably surgical diagnosis, but this will sometimes be modified or changed upon exposure of the kidney which is the immediate indication.

In chronic surgical urinary infection, the diagnosis of "chronic pyelitis" is usually made and of itself is as meaningless as the diagnosis of "laryngitis" or "cough" in pulmonary tuberculosis. While persistent pyuria may reflect only a chronic prostatitis for example, or pus in an improperly

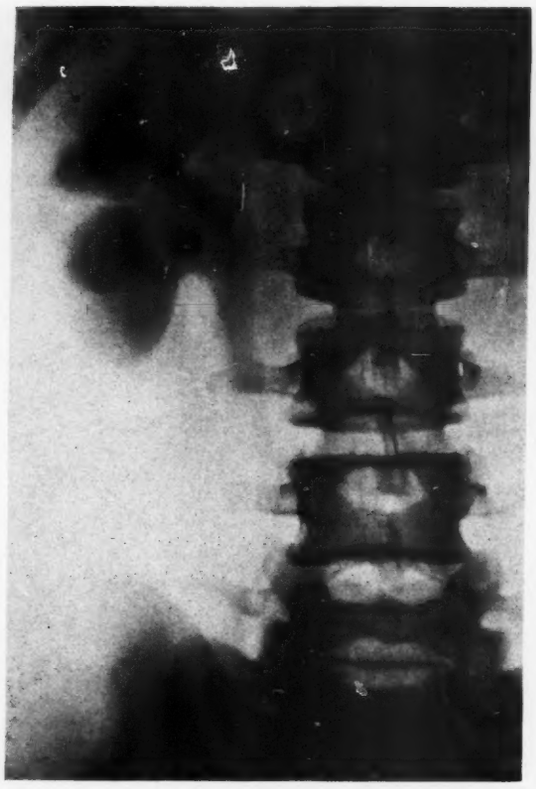


Fig. 5. Infected (tuberculous) hydronephrosis secondary to tuberculous stricture of the lower right ureter. Two and a half years previously the opposite kidney had been removed for tuberculuous pyonephrosis. There were advanced tuberculuous vesical changes. Here the pyelogram is essentially the same as might be observed with calculus obstruction of the lower ureter.

collected specimen may originate in vaginitis, the very persistence of leucocytes in the urine despite intensive medical therapy (chiefly antisepsis) demands a complete urologic examination. This exploration often discloses chronic pyelonephritis and is generally complicated by or associated with infected hydronephrosis, ureteral obstruction, renal calculus, tuberculosis or malformation. In at least half of these cases the gastrointestinal function has been deranged reflexly or by urinary toxemia and the resulting diagnosis is chronic indigestion, intestinal indigestion, chronic appendicitis, cholecystitis, gastric or duodenal ulcer and flatulence. Focusing of the attention on the secondary alimentary manifestations commonly causes the basic urinary tract disease entirely to be overlooked. Most urologic and chronic gastrointestinal surgical disease is not an emergency consideration. The patient should be given the advantage of at least an excretory urographic study when there is a doubt as to the diagnosis and before any abdominal exploration is carried out. If this is not satisfactory, an adequate urologic investigation is in order. If the urinary tract is normal, proceed with the gastrointestinal surgery. If the urinary tract is abnormal, eradicate the urologic disease first. An amazing number of patients said to have gastric or duodenal "ulcers," cholecystitis, chronic appendicitis, intestinal adhesions, and so forth, are relieved of these gastrointestinal symptoms by prostatectomy or other procedures which eradicate urinary obstruction.

INFECTED HYDRONEPHROSIS

The multiple causes of hydronephrosis are indicated in Figure 3, and the need for thorough urologic examination to identify the site and nature of the obstruction in any given case is at once apparent. In most instances the obstructive lesion is at the pelvic outlet or high in the ureter. Hydronephrosis, the commonest abdominal tumor in children, is usually congenital and is likely to be misdiagnosed as renal embryonal myosarcoma (Wilm's tumor). Mass and pain in the loin, often with hematuria, are the usual symptoms of hydronephrosis. With infection, there is pyuria. Infection added to hydronephrosis diminishes the chances of surgical renal restoration, and by the same token the need for nephrectomy is proportionately much greater in infected than in non-infected hydronephrosis. Urography, either excretory or retrograde, will demonstrate not only the gross pelvic changes but often the probable nature of the obstructive factor, e.g., stricture, calculus, aberrant vessel, kink and tumor. (Fig. 4.). In a young adult a polyp at the pelvic outlet obstructed and caused entensive hydronephrotic renal injury.

A B

Fig. 6. Solid mass of dendritic stone (calcium phosphate which filled the right kidney pelvis of a girl who was examined because of mild "chronic pyelitis" (pyuria) of three months' duration. There were no other symptoms, but spontaneous fracture of the lower third of the left femur had occurred while running six weeks previously. This strongly suggested a calcium (parathyrois) metabolic error, but neither roentgen, skeletal studies, nor blood chemistry estimation (calcium and serum phosphorus) were confirmatory. Attempted pyelonephrotomy sufficiently damaged the kidney to require nephrectomy. A, plain urogram; B, retrograde pyelogram, showing how snuggly stone fits in the calyces, pelvis and pelvic outlet.

PYONEPHROSIS

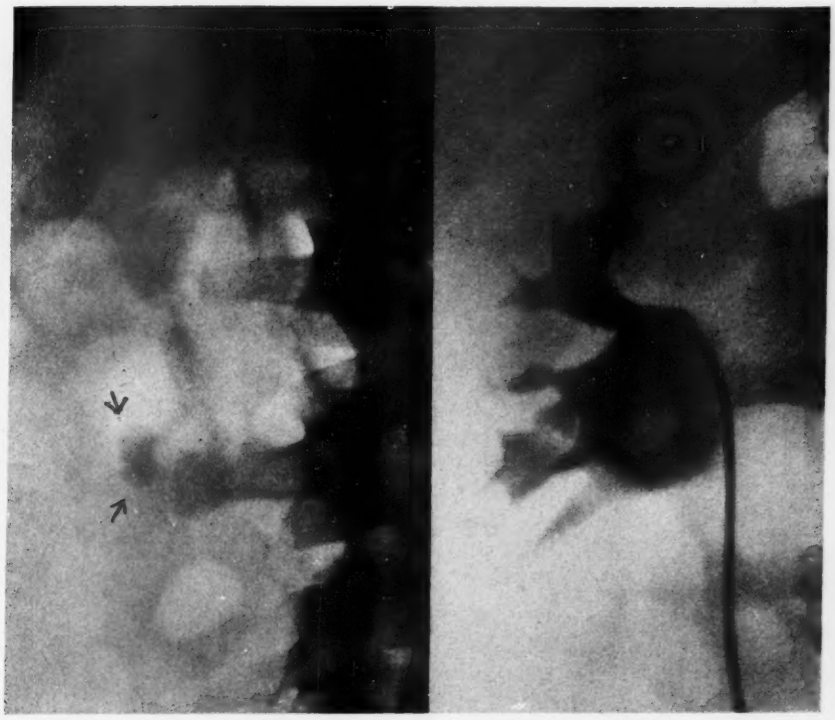
In pyonephrosis the kidney is destroyed by suppuration. This is usually the end stage of previously existing renal disease and notably infected hydronephrosis, stone or tuberculosis. Yet the infection terminating in pyonephrosis may be of sudden onset and massive, commonly caused by Staphylococcus aureus and hematogenous. In a short time the kidney is converted into a mass of parenchymal abscesses, chiefly cortical. This may become fatal if the nature of the condition is not promptly recognized and at once treated by nephrectomy. In this type the early clinical diagnosis is apt to be acute or persistent hyperacute pyelitis; in the more usual and more slowly developing varieties of pyonephrosis the usual and meaningless diagnosis is chronic pyelitis. Pyuria (often of the pea-soup variety), hematuria, pain and mass in the side suggest pyonephrosis, but the final diagnosis must rest on the urographic demonstration of the pelvic outline which frequently suggests the diagnosis of renal tuberculosis. (Fig. 5.) Yet in non-tuberculous pyonephrosis the secondary vesical inflammatory changes are meager as compared to those generally observed in the tuberculous variety. Chronic fibrotic changes in the lower ureter may render the duct rigid and its orifice held roundly open will present the so-called "golf hole." By complete urologic examination, accompanying calculus, obstruction or other disease will be demonstrated.

A B

FIG. 7. Stone pyonephrosis in a young girl. A, large stone in pelvic outlet; multiple calculi in gallbladder (picture reversed); B, enormous massive hydronephrotic destruction of kidney, secondary to calculus obstruction; nephrectomy.

TUBERCULOUS RENAL INFECTIONS

Renal tuberculosis is never primary, is predominantly a hematogenous infection and when renal and osseous tuberculosis coexist, both are usually secondary to the same pulmonary focus. Fortunately, despite improvements in diagnostic technic, the reported incidence of renal tuberculosis has definitely declined during the past twenty-five years. Yet renal tuberculosis should be suspected in any patient with so-called cystitis existing more than a month. The demonstration of tubercle bacilli in the urine practically always means renal tuberculosis. Smegma bacilli will not confuse if the specimen for examination has been properly collected. Tubercle bacilli may readily be demonstrated in the urine in 80 to 90 per cent of the cases of renal tuberculosis simply by performing the usual acid-fast (steaming carbol-fuchsin) staining of the sediment of aseptically collected and centrifuged urine. The specimen is centrifuged at 2,500 revolutions per minute for fifteen minutes. Characteristically, tuberculous urine is acid, pale, contains albumin, pus, blood (microscopic or gross) and tubercle bacilli. Often there is mixed infection chiefly with Bacillus coli.



A B

Fig. 8 A, stone of low radiopacity in right kidney pelvis. B, "negative" stone shadow in pelvis of kidney shown in A. The "negative" or vacuolization element is due to the relatively lesser radiopacity of the stone than of the radiopaque media; pyelolithotomy.

In the initial stages of renal tuberculosis, the disease picture is usually that commonly seen in chronic non-tuberculous pyelonephritis with pyuria, occasional renal pain; marked vesical irritability occurs early in over three-fourths of the patients. Painless spontaneous hematuria occurs in 5 to 10 per cent and frequently announces the disease. Its appearance signifies the rupture or erosion of a tuberculous lesion into the pelvis. Intermittent hematuria occurs in a fourth of these patients with renal tuberculosis, and is exceeded in incidence only by pain in the loin which is usually dull. Renal or ureteral colic is rare unless clot or calcareous caseous débris occludes the pelvic outlet of ureter. Fever is practically always present, low-grade, fluctuant or hectic and may reflect either the renal or the pulmonary tuberculous lesion or both. Excretory urographic studies will often suggest the diagnosis but we still prefer complete urologic studies with retrograde urography when the diagnosis is in doubt. There is negligible danger in retrograde pyelography carefully and gently carried out and No. 4 catheters are employed. Free return flow down the ureter and about the small catheter precludes pelvic overdistention by the injected radiopaque media; we employ Skiodan. Unless the pelvic outlet or upper ureter are occluded by tuberculosis débris or sclerosis, and no urographic media can get through, the pyelogram will indicate renal tuberculous fibrosis, either local or widespread parenchymal ulceration, commonly with cavitation and heavy calcium infiltration, and sometimes perirenal tuberculosis as well. The correct differential diagnosis should be made early and it is particularly important to distinguish between renal tuberculosis and chronic non-tuberculous pyelonephritis. (Fig. 5.) The prospects of cure in renal tuberculous rest largely on early and correct diagnosis and prompt nephrectomy.

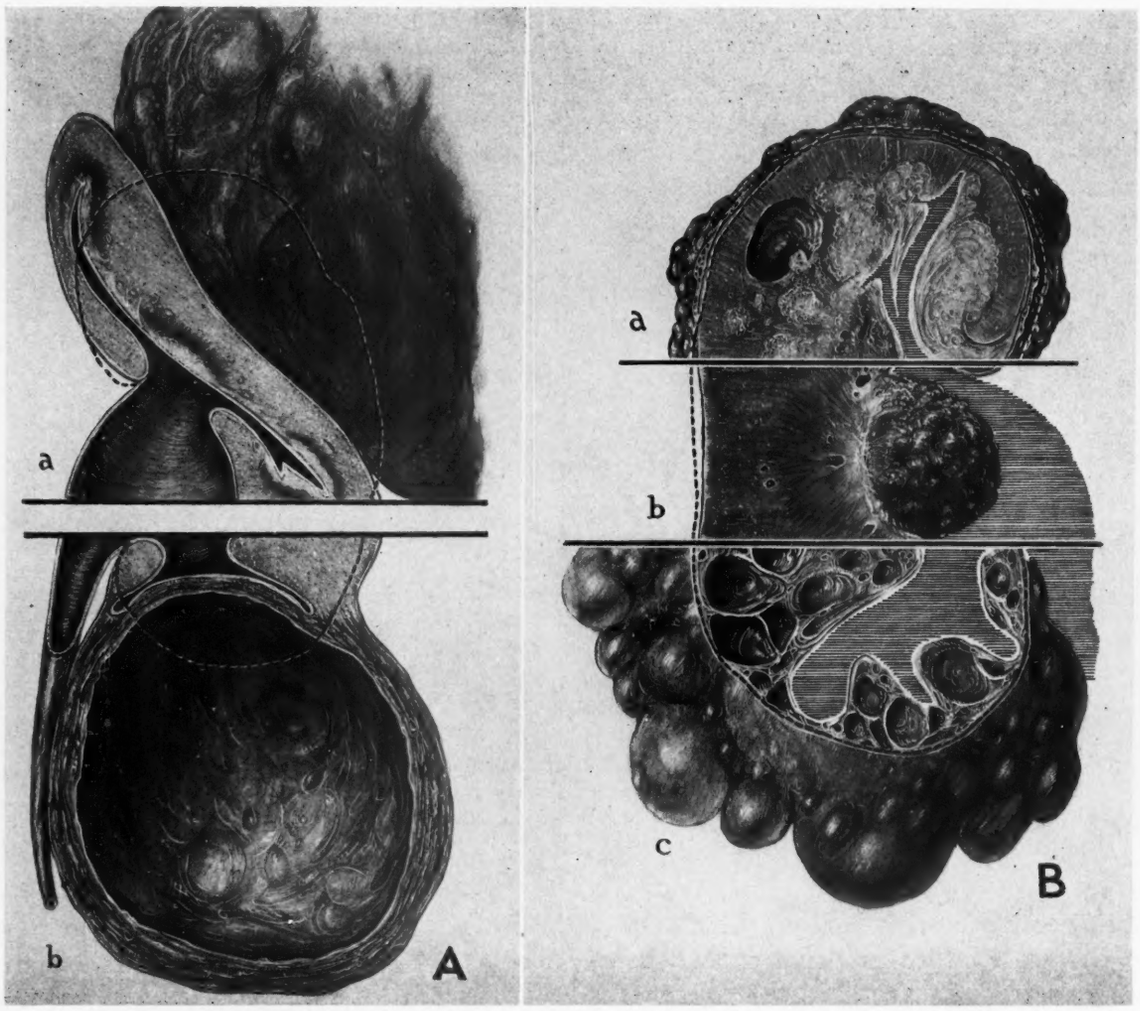


Fig. 9. Pyelographic findings in renal tumor and perirenal tumor. A, schema showing how pelvic deformity, compression or elongation results from tumors; (*a*) compressing the tumor from without or (*b*) from tumors arising within the kidney (e.g., here a cyst; see also Figure 12). B, pelvic changes in intrarenal tumors; (*a*) formation of "spider" pelvis by compression of growing parenchymal tumors; note the flattening and elongation of the calyces; (*b*) pelvic filling defect by papillomatous tumor; (*c*) bizarre pelvic irregularity and calyceal rounding seen in polycystic renal disease.

RENAL CALCULUS

Pain in the renal area or along the course of the urinary tract together with microscopic or gross hematuria and pyuria are the classic symptoms of renal calculus. The pain may be dull, intermittent or sharp colicky. Yet when the stone is large and immobile in the pelvis or is well enclosed in a renal segment, there may be no pain. This is the so-called "silent" stone. (Fig. 6.) Urography will establish the diagnosis of calculus in 90 per cent of the cases. (Fig. 7.) Pure uric acid stones cast no shadows but these and other relatively non-opaque urinary calculi can usually be demonstrated as "negative" shadows in the excretory or retrograde urogram (Fig. 8) or as "positive" shadows in a pneumopyelogram in which air is injected rather than a radiopaque media. When renal pain exists, calculus is generally considered at once but in our observation complete examination commonly discloses the primary pain-producing condition more often to be chronic pyelonephritis, hydronephrosis or tumor. Gastrointestinal symptoms and disturbances are reflexly produced in a third to a half of patients with renal calculus disease, and often cause the diagnosis of appendicitis or cholecystitis to be made. Here

needless intra-abdominal operation is a common sequel and the result of failure to perform adequate examination of the patient.

RENAL TUMOR

In an adult painless fresh hematuria most often means tumor and in a child, nephritis. A mass simulating renal tumor may be produced by a number of conditions such as hydronephrosis and tuberculosis and establishment of the correct preoperative diagnosis will largely rest upon the urographic interpretation. Here retrograde pyelography is preferable to the excretory variety since changes in the finer pelvic markings are of paramount diagnostic importance and in renal tumor these changes are too often inadequately demonstrated by the excretory method. The pelvic outline in renal tumor is usually bizarre, displaced, distorted and with compression and elongation of the calyces, the characteristic "spider" type of pyelogram is obtained. (Figs. 9B and 10.) Yet in some instances the pelvic outline (tumor obstruction) suggests simple hydronephrosis, or there may even be a filling defect in the pelvic outline caused by papillary or polypoid growth. (Fig. 9B.) The diagnosis can nearly always be made promptly and upon this and early nephrectomy hinge the future hope for the patient's recovery.

RENAL MALFORMATION

Surgically the most important of the common renal anomalies are agenesis, hypoplasia, reduplication, ectopia, fusion, cystic (solitary, multiple, or polycystic) and abnormal mobility. In all of these conditions abdominal pain, a palpable mass or abnormal urinary findings direct attention to the urinary tract. Complete physical examination must necessarily include the genitalia and here it should be noted that in one of three patients with major deformities of these organs, the upper urinary tract is also anomalous. Ureteral and pelvic reduplication is the commonest anomaly observed but in seven clinical cases of

FIG. 10. Pyelogram in renal tumor showing distortion and displacement of kidney pelvis. Here the pelves are not compressed as they commonly are found to be in renal tumors (spider type).

double uterus seen by Everette L. Campbell and me together or individually, there was unilateral renal agenesis. In four infants with double uterus seen at autopsy, there was renal agenesis and polycystic renal disease in one each and renal hypoplasia in two. The anomalous kidney is more prone to disease than a normal one. In all of these renal deformities the correct diagnosis can almost always be made by urography and when the excretory urogram is not thoroughly satisfactory and demonstrative, employ retrograde pyelography.

Congenital (or surgical) absence of one kidney should be recognized so that the solitary functioning kidney is not unwittingly removed as has been reported in a surprisingly large number of instances.

Aplasia of the kidney is of the same clinical importance as agenesis—the diminutive organ will not support life should

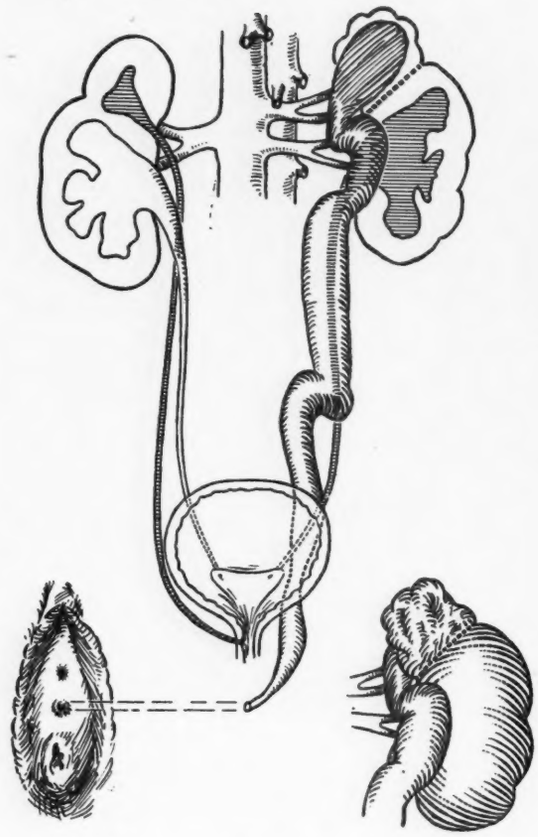


Fig. 11A. Fig. 11B.

Fig. 11. A, bilateral ureteral and pelvic reduplication with infected hydronephrosis of upper half of left kidney. Ureter from this pelvis opened ectopic in the introitus. Dotted line indicated line of renal resection. Symptoms were pain in the side and pyuria. B, pyelogram in A.

the other kidney be removed. Hypoplastic kidneys are notoriously the site of pain and require removal. Pain is believed to result from compression of nerve endings by the contracting non-functioning nephrosclerotic tissue. Pyelography makes the diagnosis. The pelvis is small, infantile, and may be of the simple calyceal variety. Occasionally, the upper portion of the ureter to the diminutive kidney has no lumen or is a fine fibrous thread; this suggests agenesis rather than aplasia. Here surgical exposure will enable the anatomic distinction to be made. Yet this is usually of academic rather than clinical importance.

Renal reduplication occurs about once in 150 individuals. In most instances the malformation is of no moment but occasionally half of the organ will be diseased by calculus, obstruction, suppuration and so forth. When this is demonstrated by urography, resection of the kidney with removal of the diseased segment will usually leave sufficient renal tissue to support life should the other kidney subsequently be lost. A satisfactory pyelogram is of utmost value to the surgeon as it will indicate the best line of incision with which to divide the kidney and not enter the pelvis. (Fig. 11.)

Ectopic kidneys are most often found in the lower pelvis where they are readily palpable and are likely to be thought to be abdominal or pelvic tumors. This has been the diagnosis in the several pelvic ectopic kidneys I have seen. Urography

establishes the nature and rotation of the ectopy and will indicate to the surgeon the length and course of the ureter should surgical elevation be desired.

Fused kidneys may assume bizarre shapes; the best known is the horse-shoe kidney in which there is either lower or upper polar fusion or complete fusion into one single mass. (Fig. 12.) Sometimes the lower pole of one kidney is fused with the upper pole of the opposite ectopic organ into S (sigmoid kidney) (Fig. 13) or L shapes. These fused kidneys are notoriously prone to disease, especially stone and infection, and since treatment and the resectability of the diseased segment will largely be indicated by urologic examination, a satisfactory pyelogram is of the utmost importance. A retrograde pyelogram is preferred. Abdominal pain, especially about the umbilicus, is often observed in abnormally formed kidneys, especially in horse-shoe kidney disease.

In *solitary cystic disease*, a dull ache in the side is the principal symptom and a mass in the loin is the usual finding. The pelvic outline as demonstrated by pyelography may be normal but it generally suggests tumor with compression of the kidney pelvis. (Fig. 14.)

In *polycystic renal disease*, the initial symptoms too often cause only the diagnosis of chronic interstitial nephritis to be made until or unless hematuria and pain in the loin occur or the bilateral renal masses are discovered. The pyelogram shows bizarre irregularity with a notable tendency to crescentic dilation of the major calyces. (Fig. 9B.) Sometimes the cortical cysts can be palpated through the abdominal wall.

The *abnormally mobile kidney* is likely to cause endless symptoms in nervous patients (renal pain, palpable mass, reflex gastrointestinal disturbance with pronounced psychoneurotic manifestations), but calls for no treatment unless there is ureteral obstruction. A good pyelogram is, therefore, of utmost value not only to make the diagnosis but to conclude whether nephro-ureterolysis and renal suspension are indicated. The urograms are made with the patient in the Trendelenburg position as well as upright in order that the degree of mobility of the kidney can be observed. To repeat, unless there is obstruction, operation should be withheld.

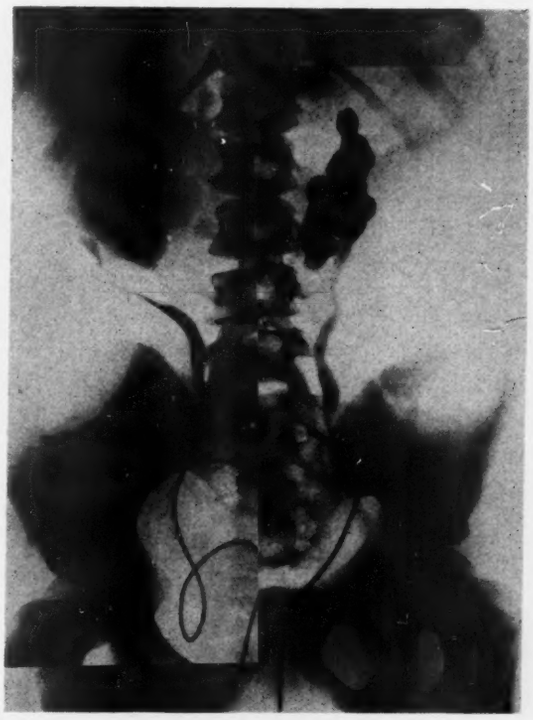


FIG. 12. Horse-shoe kidney disease in twelve-year old girl who, because of persistent pain in the right upper quadrant, had been treated for several months under the erroneous diagnosis of gallbladder disease. During this time she had had eight cholecystographic series and three complete gastrointestinal radiographic studies. Excretory urography suggested the renal anomaly which was proved by retrograde pyelographic study (composite pyelogram shown above). Following resection of the horseshoe kidney through the isthmus with removal of the right half, the patient was entirely relieved of all symptoms, her digestion became normal and she gained weight. This case splendidly illustrates the close symptomatic interrelation between gastrointestinal and urinary tract disease.

SUMMARY

From the foregoing it is readily noted that accurate preoperative diagnosis can be made in surgical renal disease and here pyelography is indispensable. By recognition of (1) the high incidence of urologic disease as the cause of abdominal symp-

FIG. 13. Crossed fused renal dystopia in girl examined because of chronic pyuria and intermittent pain in the right loin. During an acute attack of the latter, a normal appendix was removed. The urogram indicates a variety of pelvic misdirection commonly observed in this condition. There is some dilation of the ureter which drains the upper renal pelvis (congenital ureterovesical junction stricture). This stricture readily responded to therapeutic dilation.

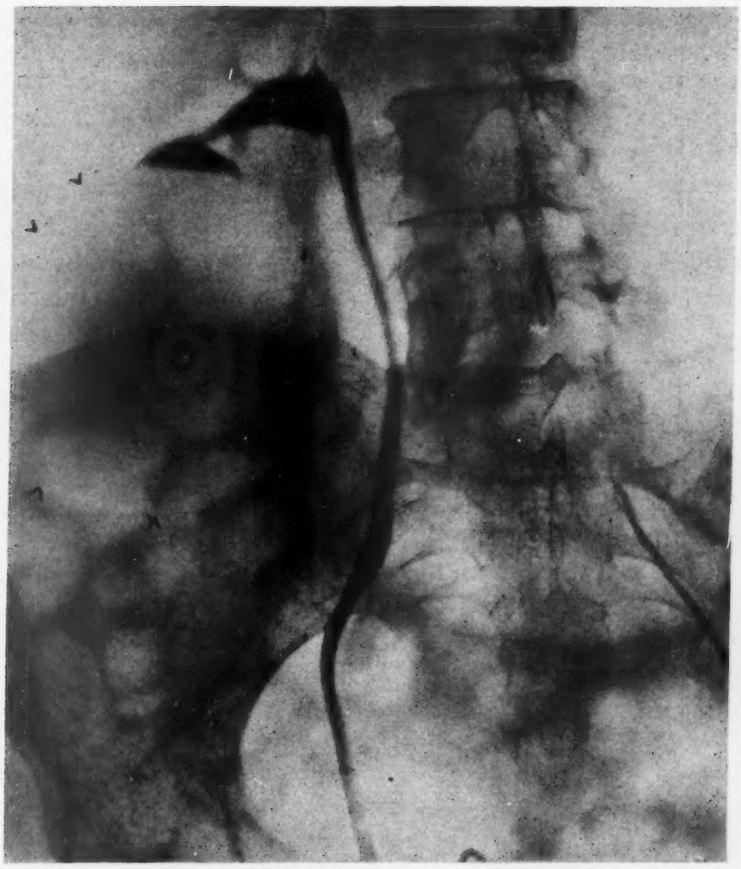


Fig. 14. Large solitary renal cyst involving the lower pole of the right kidney. There is marked pelvic distortion and compression with transverse flattening of the lower calyx (compare Figure 9A). Excision of the cyst with scissors with complete removal of the cyst lining is simple, curative and conserves the remainder of the kidney.

toms, (2) the facility with which the renal diagnosis commonly can be made, it is at once apparent that no patient with vague and indistinct abdominal pain should be subjected to laparotomy without at least the benefit of a satisfactory excretory urographic study. If this dictum is recognized, more cases of "abdominal adhesions," "chronic appendicitis" and the like will become bona fide instances of renal or ureteral disease and usually will be characterized by obstruction.

ACUTE SURGICAL LESIONS OF THE PELVIS*

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UNDER this topic I would place six main headings and attempt to discuss them without going into too much detail.

Acute Salpingitis. This may or may not be of specific origin but usually is specific and is due to Neisserian infection. The status of the treatment of this disease has almost completely swung from a surgical approach to a medical one, especially since the introduction of the sulfa drugs and the more recent, almost specific, penicillin. There remains, however, about the same relative number to be treated surgically as were treated medically fifteen years ago, which is almost a complete reversal of trends. That, of course, is all to the good as regards the patient. It certainly was not pleasing to the surgeon to have to make it impossible for conception to take place for so many female patients in their early twenties. Now the tables have turned. Someone has said gynecologic surgery is on the way out.

The diagnosis of this disease is usually not difficult, especially if a careful history is taken, and uterine cervical and Skene's gland smears are made. One must also bear in mind that most acute infections of this kind are apt to become active immediately after the menses. Surgery is only indicated after rest and the free use of the sulfas and penicillin. The surgical indications need only *then* be faced and conservative surgery would be dependent upon the social status of the patient.

Extra-uterine Pregnancy. This presents problems in diagnosis and therapy which are often very difficult. A clear classical case in which there is a history of the patient's being one to four weeks overdue in menses, with every other indication of pregnancy, then sudden acute abdominal pain with distress in the pelvis and frequently pain in the diaphraghmatic area, is not difficult of diagnosis. But when even these symptoms occur before menses are due, or at the time menses are due, they present problems sometimes hard to evaluate; but, with definite pain, signs of hemorrhage and increased white count, one is justified in opening the abdomen for exploratory purposes. Even during exploration the signs may be so scant as to present a real problem, that is, when a very early pregnancy is attached to an ovary. A more difficult problem is to differentiate between a ruptured extra-uterine pregnancy and a tubal abortion. If the latter is present, surgery may not be indicated. I am sure many such extra-uterine abortions have healed of themselves without surgery.

Ruptured Corpus Luteum Cyst. Ruptured ovarian cysts may be divided into two types: the hemorrhagic and the ruptured simple cyst. In the hemorrhagic type, the symptoms are largely those of severe shock with signs of hemorrhage, and are often hard to differentiate from those of extra-uterine pregnancy. The symptoms of rupture of the simple corpus luteum cyst are often confused with those of acute appendicitis. There are several points to be emphasized, however. The pain of a ruptured cyst is very severe and sudden, often occurring on a shopping tour or card party. The history should tell one whether the time of sudden pain is about midway between menstrual periods. Occasionally, the patient is operated upon at once by the "eager" operator, only to find, instead of a ruptured appendix, an accumulation of a straw-colored fluid in the pelvis or a ruptured corpus luteum cyst. For-

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tunately, acute hemorrhage is seldom present. Greater care in eliciting a history will save the surgeon some embarrassment.

Acute Appendicitis. One should be able to differentiate its symptoms from those of other acute pelvic lesions except when the appendix attached to a loose low-lying cecum (often seen in patients with enteroptosis) lies posterior to the uterus or in the cul de sac of Douglas. In this type of case one may find an acutely inflamed appendix, even a ruptured one, lying in the cul-de-sac of Douglas and resembling on examination the findings of acute salpingitis, except that the inflamed tumor mass is not bilateral as in most cases of acute salpingitis. Fortunately, nature is usually good to such a lesion by walling off the appendix, and if the diagnosis plus surgery are delayed, no hazard is added by the delay. If one should encounter pus, drainage through the cul de sac is readily and safely done.

Twisted Ovarian Cyst. This alone or complicated by other chronic pelvic conditions, will at times present a problem, and again a delay of twenty-four to forty-eight hours in making a diagnosis entails no undue hazard to the patient. Surgery, of course, is always necessary.

Necrotic Myoma of the Uterus. Acute necrosis of a myoma of the uterus or the twisting of a pedicle of a myoma presents a diagnostic problem, even though signs of local tenderness may not be present; but surgery is indicated eventually and offers a safe and prompt solution.

There are other pathological lesions in the pelvis, but of such rare occurrence that they will not be mentioned. They so often complicate the main problems just mentioned, that surgery for one condition will remedy the other.

Thus one sees that there are many real challenges in making an accurate diagnosis of acute conditions in the pelvis. Certain it is that the advent of the sulfonamides and penicillin have averted surgical intervention and saved many a reproductive organ from a trip to the pathologist's table. A detailed history of premonitory and immediate symptoms is of the greatest value in a proper diagnosis. The technical details of surgery present no great problem to the average surgeon.

THE TREATMENT OF ACUTE PERFORATION OF DUODENAL ULCER

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THIS communication records the details of the management of 130 consecutive cases of acute perforation of a duodenal ulcer admitted to the First Surgical Division of the Toronto General Hospital since July 1, 1929. This critical analysis has confirmed the soundness of the previously published principles (1)[2] which have guided our therapeutic procedures in the management of this grave emergency. This study excludes all cases of perforated gastric ulcers. Our experience with such emergencies has been small, operation in many instances revealed that the perforation had occurred through a gastric carcinoma. A subsequent presentation is planned to assess the ultimate fate of this group of perforated duodenal ulcers. We concern ourselves at the moment with the management of the emergencies.

TABLE 1

Total Cases	130	D	13
Cases No. 57 & No. 98	2	refused operation	
Cases No. 104 & No. 128	2	moribund—no operation	
Case No. 76	1	undiagnosed	
Total not operated	5		
Total No. of operated patients	125	D	8 = 6.4 per cent

Deaths

1. Case No. 48—Pulmonary embolism. Autopsy 10th day
2. Case No. 67—Pneumonia
3. Case No. 79—Pneumonia + Senility
4. Case No. 72—Pneumonia + Mild peritonitis
5. Case No. 78—Uraemia
6. Case No. 80—Unrecognized pelvic abscess—perf., general peritonitis
7. Case No. 102—Subphrenic—undiagnosed—perforated to bronchus
8. Case No. 126—Multiple pulmonary abscesses—Subhepatic abscess—Chronic Osteomyelitis Femur + Pyelonephritis.

A reference to Table 1 will show that of this total group of 130 cases there were thirteen deaths. Five patients were not operated upon; two refused operation; two were moribund on admission and their condition could never be improved to a point where it was justifiable to advise operation; one patient died before the diagnosis was made, despite the fact that he was in the hospital for two weeks. At autopsy it was found that an unrecognized left subphrenic abscess had perforated into his bronchus.

One hundred twenty five patients were operated upon with eight deaths, an operative mortality of 6.4 per cent. The details of the deaths are presented in Table 1. Three predominant areas are responsible for the fatality, chest, subphrenic space and the pelvic cul-de-sac. There were three patients in whom pneumonia was a factor responsible for death. In one it was the sole factor, in a second it was part of a general senility and in a third it was complicated by a mild peritonitis. It was considered at autopsy that the actual cause of death was the pneumonia, as the peritoneal reaction was minimal; two patients had undiagnosed subphrenic abscesses. One perforated into a brochus similar to that which occurred in the non-operated undiagnosed case. The other case presented multiple pulmonary abscesses, multiple intraperitoneal abscesses, pyelonephritis, as well as a subphrenic abscess. This patient had a coincidental chronic osteomyelitis of the femur. While it is impossible to prove irrefutably that the widespread infection was metastatic from the chronic discharging osteomyelitis, this seems the most logical explanation of the dissemination of his infection. One patient died of a pulmonary embolus on the tenth day; autopsy showed that the operative site was normal.

We are impressed by the infrequency of emboli and peripheral vascular accidents postoperatively since we have been practising early ambulation. It is too soon to be absolutely certain of the rôle which early ambulation is playing in the diminution of postoperative vascular accidents. It is a fact that coincidental with this practice there has been a very sharp reduction in the incidence of these complications. During this same period extreme care has been taken in the preoperative and postoperative management of our surgical patients, particularly in regard to the correction of their nutritional and biochemical defects. This management, as well as early ambulation, is a contributing factor to the diminution of the postoperative complications.

It is interesting to note the age incidence. There were seventy-two patients aged fifty years or over and fifty-eight were forty-nine years old or younger, with an average age of forty-eight and one-half years. However, in the wide spread of the age group, the youngest patient was sixteen years of age and the oldest was eighty-three, so that age as a factor in the diagnosis is perfectly useless. It is possible, however, to have helpful information by considering the ratio of males to females. There were 123 males and seven females in the group. This fact has warned us against the snap diagnosis of a perforated duodenal ulcer in women. When we suspect this as a possible diagnosis, the infrequency of its occurrence is sufficient to demand that we canvas every other alternative diagnosis before accepting it as the cause of the patient's illness.

The diagnosis of an acute perforation of a duodenal ulcer is not difficult in most cases, nevertheless we have made errors. These errors have arisen from the difficulty in differentiating an acute perforated ulcer from acute appendicitis, acute pancreatitis and acute cholecystitis, in diminishing order of frequency. Only a small percentage of the patients with an acute perforation of a duodenal ulcer fail to give a history of previous gastrointestinal distress. If a patient whose signs and symptoms present diagnostic difficulty has had a long continued indigestion of the duodenal ulcer type, with major and minor periodicity, it is surprising in what a large percentage of these patients there has been an acute exacerbation in the intensity of their distress just prior to the perforation. This is not true in patients who suffer from acute appendicitis, acute pancreatitis or acute cholecystitis.

The estimation of the serum amylase in all patients who suffer from indefinite, sudden abdominal pain we have found to be of great value in confirming or excluding the presence of acute pancreatitis. We have found invariably in all cases of acute hemorrhagic pancreatitis that the serum amylase has been elevated. This finding has reduced the incidence of errors and confusion in the diagnosis of acute pancreatitis.

Atypical appendicitis, however, presents the greatest diagnostic confusion. One patient was operated upon five hours after the onset of the pain, diagnosed as a perforated duodenal ulcer. He had a perforated appendicitis in an imperfectly descended cecum, the appendix lying in contact with the duodenum. The character of the original pain in acute perforation of a duodenal ulcer is of value in differentiating this emergency from extraperitoneal lesions with which it may be confused. A sudden coronary occlusion is often associated with very severe abdominal pain, but this gradually increases in severity, and permits the patient to reach a place where he can comfortably rest; whereas a patient who has a sudden perforation of a duodenal ulcer is as a rule suffering a pain so sudden, severe and crippling as to immobilize him for a varying period of time, often as long as an hour. The coronary pain is usually persistent, or increased in a crescendo, while the pain of a perforated duodenal ulcer usually diminishes until at the end of one or two hours there occurs the tragically dangerous socalled "latent period," in which the patient is reasonably comfortable while lying still, and pulse and temperature are normal. However, on physical examina-

tion true rigidity of the abdominal muscles is always present. This true rigidity is differentiated from the false rigidity due to fear or extraperitoneal lesions by the fact that in true rigidity the tension in the muscle is constant during the entire respiratory cycle, whereas if it be a voluntary or false rigidity there is a momentary relaxation at the end of inspiration. This finding is tremendously important. True abdominal rigidity as an accompaniment of an acute hemorrhagic pancreatitis is extraordinarily rare. If in addition to the abdominal pain there is a pain referred to the shoulder tip, we have evidence of diaphragmatic irritation, which can be confirmed by a flat *x*-ray plate taken in the erect position, which will show the presence of gas under the diaphragm. While gas is not always present under the diaphragm, it is of inestimable diagnostic value when demonstrated by *x*-ray examination. The absence of gas under the diaphragm is of no diagnostic value. It was not noted in seven consecutive cases of perforated ulcers. The loss of liver dullness as elicited on physical examination by percussion of the abdomen, may be misleading. We have found that the normal area of liver dullness may be obliterated by distension of the colon. Hence the absence of this area of dullness does not necessarily indicate that free gas is present in the peritoneal cavity.

Until May of this year we had never seen a patient suffering from a perforated duodenal ulcer who did not have a leucocytosis. This patient on admission had a white blood count of 3.4. On the fifth postoperative day it had risen to 4.0 and on the sixth postoperative day was 11.2. The bacterial study of this exudate showed it to be the result of a pure culture of pneumococcus. A previous communication[3] has stressed a trinity of signs and symptoms as indicative of intraperitoneal irritation. The acceptance of this trinity as evidence of an intraperitoneal abnormality has become increasingly justifiable with its increasing use. The trinity is (1) The association of pain and tenderness. If the patient complains of pain without being also tender in the same area, the lesion is extraperitoneal. This is the most valuable single evidence in differentiating intra- from extraperitoneal lesions. (2) One point of maximum tenderness. The patient will put his finger on a point rather than place the whole hand over an area to indicate the site of his distress. (3) It hurts the patient to roll over in bed. If in a patient who is complaining of abdominal pain, this trinity of signs and symptoms is present then such a patient has an abnormality within the peritoneal cavity. This trinity may occur as the result of free blood in the peritoneal cavity; excluding this it is due to an acute inflammatory lesion.

If in association with this trinity rebound tenderness is present, it is inconceivable that the patient has a normal peritoneal cavity. Rebound tenderness is elicited by suddenly releasing the palpating hand which had been pressed firmly on the abdominal wall. This sudden release of pressure is accompanied by sudden abdominal pain if rebound tenderness is present

If after a careful assessment of the data elicited by history analysis, physical examination, *x*-ray and laboratory studies, the diagnosis is still in doubt only as to the cause of the intraperitoneal catastrophe, the site of the incision must be determined. Under such conditions we always explore the peritoneal cavity through a gridiron incision in the right iliac fossa. This incision is atraumatic and harmless and almost invariably will solve the diagnostic problem. Should the lesion be an acute appendicitis, the operation may be performed through it. If on the other hand, the patient, should be suffering from a perforated duodenal ulcer and not from appendicitis, it is important to recognize and differentiate a peri-appendicitis from a true appendicitis.

Following the acute perforation of a duodenal ulcer, the inflammatory exudate and duodenal content flow down along the right colic gutter, resulting in the pro-

duction of an inflammatory reaction about the appendix. However, to recognize this type of reaction as a possibility is sufficient to avoid an error in mistaking the local lesion as being the result of a true acute appendicitis.

It is obvious that under such circumstances a second incision is imperative to give access to the perforated ulcer. It is not necessary to apologize for this. Such a sequence is infinitely safer, less traumatic and accompanied by fewer postoperative hernias than if one explored the duodenum for a suspected perforation and then had to enlarge the incision sufficiently to enable the removal of an acutely inflamed appendix.

Having accepted the diagnosis of an intraperitoneal catastrophe, one then must define the surgeon's responsibility in an emergency abdominal operation. The only justification for doing an emergency abdominal operation is that to defer such a procedure would endanger the patient's life. Thus many operations will have to be undertaken at inopportune times in an inadequate environment, with the staff and facilities available: therefore, the surgeon who is readily accessible to perform the operation is the best surgeon to close an acute perforation of the duodenum. The operation which will be proposed is so simple that it can be done by any medical man who has had even a modest training in surgical technic. There is no justification for debating the wisdom of a gastroenterostomy or a gastric resection. As a corollary, it is unwise to transport a patient suffering from an acute perforation of a duodenal ulcer over long distances if there is anyone available locally who can carry out the necessary operative procedure. The operation should be one that confines itself solely to dealing with the lesion responsible for the emergency and dealing with it efficiently in the most simple manner. It is obvious, in a patient who requires operation for an acute perforation of a duodenal ulcer, that the prime requisite is the closure of the perforation. We must, therefore, answer the question: What closes all perforations in the gastrointestinal tract, even those that are inevitably made in the performance of abdominal operations requiring gastrointestinal anastomoses? While theoretically a needle may be inserted in the gastrointestinal tract in the performance of anastomoses without entering the lumen, this probably rarely occurs. If one could examine microscopically the course of the needle and its suture, it would frequently transverse mucous membrane. However, despite this, successful anastomoses are possible because as a result of even the minimal trauma of such a suture, there is sufficient tissue damage to produce fibrin ferment, which will result in the formation of fibrin from the inevitable inflammatory exudate which accompanies all wound healing. Thus we must remember that fibrin is the substance which seals all perforations in the gastrointestinal tract. Therefore, any operation which will result in the gross production of fibrin would be an ideal operation to utilize in closing a perforated duodenal ulcer.

Having accepted this philosophy of the surgeon's responsibility in the performance of an emergency abdominal operation, an oft-quoted aphorism states, "The sun must never rise nor must the sun ever set before a patient who is suffering from an acute perforation of a duodenal ulcer is operated upon." This statement is completely erroneous. It is founded on the belief that the patient is always sick because of a bacterial peritonitis. This is incorrect in almost all instances. In sixty-five patients on whom culture was taken, thirty-four were sterile; thirty-one showed organisms of various kinds and only six grew pathogenic organisms from the peritoneal exudate. One was a pure culture of typhoid;[4] one Streptococcus hemolyticus; one penumococcus and only three grew colon bacilli. That infection is not such a "bete noire" as is commonly believed is supported by the fact that of the eight operative deaths only four, or 50 per cent of these deaths had an associated peritoneal infection, and indeed

in two of them the peritoneal infection probably was a minimal factor in their terminal illness.

What then is responsible for the disastrous clinical state in which we find these patients? The following case is typical of many, and supports the thought that pain, nutritional defects and biochemical imbalance are the determining factors in the patient's clinical state:

A male, aged fifty-five, was admitted to the Department of Medicine for investigation of an indigestion which previous admissions had failed to identify as due to structural disease. He previously had had many operations and recently had complained of frequent abdominal pain. During his hospitalization for investigation, at 6 A.M. he suffered sudden epigastric pain associated with abdominal tenderness and muscular rigidity. When seen in consultation he was desperately ill; his blood pressure was 75/50. The diagnosis of a serious intraperitoneal catastrophe was made most likely the result of an acute perforation of a duodenal ulcer. *X*-ray showed no free air under the diaphragm. His general condition was so poor that immediate operation was considered unwise. He was placed in an oxygen tent, pain was relieved by sedatives, and intravenously he was given by the drip method 1500 cc. of saline and glucose, as well as 500 cc. of plasma and 500 cc. of whole blood. At the end of ten hours his blood pressure was 110/76 and his pulse 80. His appearance and general condition were in marked contrast to that when first seen. At operation a perforated duodenal ulcer was found, and *cultures taken from the intraperitoneal exudate were sterile*, despite the fact that operation was not undertaken until sixteen hours after the perforation, ten hours of which were consumed in the preoperative preparation. He made an uninterrupted convalescence.

This case is not unique in the series, but specifically presents evidence which refutes the thought that a bacterial peritonitis is responsible for the serious clinical state which is so often an accompaniment of an acute perforation of a duodenal ulcer.

A plea is made for a careful assessment of the patient's nutritional state and biochemical balance before any operation is undertaken in cases of acute perforation of a duodenal ulcer. During the years in which this attitude has guided our preoperative management of such patients we have become convinced of the soundness of this regimen. The transformation of a seriously ill patient from what appeared to be a desperate if not a fatal illness to a clinical state where operation could be safely undertaken represents one of the thrills of a surgical career.

Thus we accept the following criteria as sufficient to justify delaying operation in order to improve the clinical state: (1) A severe nutritional disturbance, which can be gauged by the diet taken immediately preoperatively; (2) severe dehydration, (3) severe anemia and (4) a low blood pressure, rapid pulse, severe abdominal pain, as a part of the general picture of shock. Patients now, instead of being admitted directly to the operating room are always sent to the ward, where their clinical state is assessed carefully. It is surprising how the intravenous administration of fluids, with heat, sedation and oxygen, will improve most patients in a very few hours. We have never regretted the time consumed in this preoperative preparation, despite the fact that we are very conscious that it is deferring the operation.

The operative procedure, which is in most instances performed by the resident or senior interne, is carried out under spinal anesthesia. This type of anesthesia we are convinced has great advantages over the inhalation anesthesia. The abdomen is still, in marked contrast to the plunger action of the diaphragm and liver in deep inhalation anesthesia, which action has been shown by Gordon to disseminate the intraperitoneal exudate. Gordon[5] in a series of dogs, introduced India Ink into an area in the peritoneal cavity. One group was anesthetized with spinal anesthesia. The second group was anesthetized with inhalation anesthesia. The dissemination of India Ink throughout the peritoneal cavity was infinitely more diffuse in the latter than in the former group.

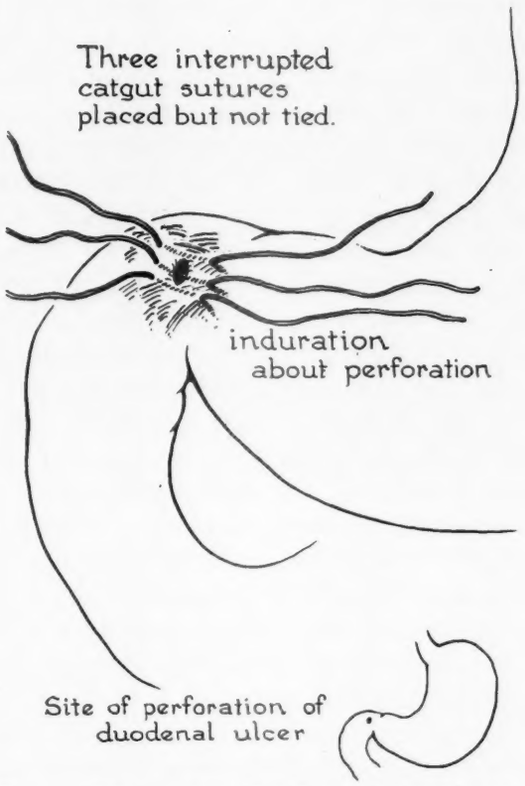

FIG. 1. The sutures placed in relationship to perforation.

FIG. 2. Sutures tied holding omental graft in position. They are tied only tightly enough to hold the graft with no attempt to approximate the sides of the perforation.

The basis of our operative procedure is the gross production of fibrin. Traumatized tissue will liberate the necessary ferments to stimulate the production of fibrin from an inflammatory exudate. On many occasions when operating for chronic duodenal ulceration, omental or fat tags were adherent to the base of a duodenal ulcer. This we interpreted as a control of minimal perforation. For many years we had used omental and fat grafts to cover raw areas of bowel in secondary operations and to close the ureter and common bile duct after the removal of calculi. It seemed logical that such a maneuver would adequately seal the perforation of the duodenum. Repeated operations have proven that such is true. We aim now to place a piece of adjacent fat or omentum attached or free over the perforation and hold it in place with sutures.

The abdomen is opened through the right rectus muscle and the fibrin leads the surgeon to the site of the perforation. Two types of duodenal ulcers perforate, one, the acute variety, with a cleanly punched out hole in the duodenal cap, with no induration about it. The other is a perforation in a rigid mass of scar tissue in and about the duodenal cap. While it might be possible to close the acute ulcer, all have experienced the futility of attempting to close by suture the perforated indurated ulcer. It can, however, be efficiently closed. Three sutures are placed parallel to the long axis of the duodenum, one above, one across and one below the perforation. (Fig. 1.) A piece of fat or omentum free or detached is placed over the hole and the sutures are tied only tightly enough to hold the fat or omentum in position, with no thought that the tied sutures will approximate the margins of the perforation. Within an incredibly short time the area is covered with a thick layer of fibrin. During the war ample opportunity was available to see how rapid and how gross was the formation of fibrin about penetrating wounds of the gastro-

intestinal tract. Lewis[10] has shown that the intraperitoneal exudate in these cases contains trypsin. For this reason the excess exudate may be removed with a sucker. However, it is not necessary to be so vigorous in its removal as to prolong the operative procedure unduly or traumatize the peritoneal contents.

In view of the bacteriological studies of the intraperitoneal exudate, the abdomen is closed without drainage. We are impressed with the efficacy of using No. 32 stainless steel wire as suture material. Interrupted figure-of-eight sutures as advocated by Jones[7] are used. The patient is returned to bed with an indwelling Levine tube inserted through the nostril, and if seriously ill is placed in an oxygen tent for twenty-four to forty-eight hours. Fluid is allowed by mouth in volume of one ounce at a time every half hour if the patient desires it. Fluid and chemical balance is maintained by the intravenous administration of water, salt, glucose, plasma or whole blood as indicated. The adequate and easily recorded control of fluid balance is made possible by the use of a fluid balance sheet. (Fig. 3.) In the immediate postoperative period the edema about the ulcer obstructs the pylorus and unless great care is taken to replace fluids and electrolytes aspirated from the stomach the patient can rapidly reach a state of hypochloremia, or even go into gastric tetany. However, many complications are avoided if the stomach is kept empty with the indwelling Levine tube and fluid and chemical balance is maintained.

POSTOPERATIVE MANAGEMENT

The immediate convalescence of these patients has been delightfully uneventful, with rare exceptions. In this whole group we have had two duodenal fistulas. These can be dealt with safely and adequately, using Potter's technic.[8] The last case occurred in the patient previously discussed who had the low leucocyte count of 3,400 at the time of operation and whose peritoneal exudate showed a pure culture of pneumococci. This combination was a definitely contributing factor in the formation of this fistula. The principle of Potter's technic is based on the fact that if one can acidify the alkaline duodenal secretion and provide for its disposal on the abdominal wall, there will be prompt healing and with a minimum skin excoriation. To accomplish this a small catheter is placed as far into the fistulous tract as possible and N/10 hydrochloric acid is dripped through this catheter at the rate of approximately 25 cc. an hour. A further tube is placed just at the fistulous opening in the skin, and suction applied. Gauze soaked in 10 per cent commercial peptone then is applied as a dressing around the fistulous opening, and this provides a substitute for the skin to be digested by any unneutralized ferments. The last fistula was healed within ten days from the instituting of this management.

Three areas should always be canvassed carefully if the patient's postoperative progress is not satisfactory: (1) the chest, (2) the pelvic cul-de-sac, and (3) the subphrenic area. One patient we probably could have saved had we done a rectal examination with greater diligence. We would have recognized a pelvic abscess, which subsequently ruptured into the peritoneal cavity. We have in all abdominal cases made it a rule for years always to pass a Levine tube and always do a rectal examination on every patient whose postoperative course is causing us the slightest concern. The assistance of our medical confreres is invaluable in the diagnosis of subphrenic space infection. Even when one suspects it, it is a most difficult diagnosis, as has been shown by Warner and Doidge.[9]

There is one pitfall in the diagnosis of subphrenic space infection. If during the investigation of such a patient *x*-ray studies show gas under the diaphragm, one should not accept this as evidence of a subphrenic abscess if the patient were operated upon with a spinal anesthesia. Lewis[6] has shown that in all patients whose abdomen is opened under spinal anesthesia

FLUID BALANCE SHEET

The data indicated to be recorded on all cases having an indwelling duodenal or Miller-Abbott tube or to whom an intravenous solution is being given. All amounts to be recorded in cubic centimetres. All totals, all losses or gains, and all Transfusion Volumes to be recorded in red ink. Indicate any reaction by the letter "R" in red ink in the appropriate horizontal column under "intravenous."

FORM 260 BACK

NAME WARD OPERATION

DATE												
	7 A.M.–7 P.M.	7 P.M.–7 A.M.	7 A.M.–7 P.M.	7 P.M.–7 A.M.	7 A.M.–7 P.M.	7 P.M.–7 A.M.	7 A.M.–7 P.M.	7 P.M.–7 A.M.	7 A.M.–7 P.M.	7 P.M.–7 A.M.	7 A.M.–7 P.M.	7 P.M.–7 A.M.
Fluid Intake Per os.												
Intravenous Saline												
5% glucose in normal saline												
5% glucose in distilled water												
Blood												
Plasma												
Total intake												
Fluid Output Fistula												
Stool												
Tube						A	A					
Vomitus												
Urine												
Total output												
Total Intake Gain												
Loss												
Gastric Intake Gain												
Loss												

FIG. 3.

air is locked under the diaphragm and it may persist for as long as six weeks.

CONCLUSIONS

1. A series of 125 consecutive operations for acute perforation of duodenal ulcer is presented, with eight deaths, a mortality of 6.4 per cent.

2. The cause of the patient's serious clinical state following perforation of a duodenal ulcer is not the result of a bacterial peritonitis, but rather the result of nutritional and biochemical imbalance associated with the phenomena of shock.

3. Adequate correction of these disturbances before undertaking the operation contributes the greatest safety factor in decreasing the mortality of this catastrophe. We have never regretted the time consumed to achieve this.

4. An emergency abdominal operation should deal in the most simple manner solely but effectively with the lesion responsible for the emergency. Gastroenterostomy and gastric resection have no place in this management.

5. Fibrin is responsible for the closure of all gastrointestinal perforations. A free or attached omental graft held in position over the perforation by three sutures results in the gross formation of fibrin to close the perforation.

6. Drainage of the abdomen at the time of closure of the ulcer is unnecessary.

7. The intraperitoneal use of sulphanilamides appears to be superfluous.

8. Spinal anesthesia is the anesthesia of choice.

9. Should a postoperative fistula occur, Potter's technic is efficient.

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THE SURGICAL TREATMENT OF CHOLECYSTITIS*

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FROM the surgical standpoint, cholecystitis may be properly divided into two phases, according to the pathologic process: (1) The chronic phase and (2) the acute phase. The present discussion will be devoted to a consideration of the most salient features of the disease, particularly in relation to these two phases.

Chronic Cholecystitis. By far the majority of patients with this type may be successfully treated by medical measures. The question is: At what point does surgical treatment become necessary for the remainder? Aside from the mortality, which is comparatively low, approximately 15 per cent of patients with chronic cholecystitis are unimproved or made worse by operation. These poor results may be attributed largely to errors in the selection of patients for operation, especially those who have the noncalculous form of the disease. Many errors might be avoided by observance of more rigid criteria for surgery, and this in turn is made possible by more penetrating inquiries into the patient's complaints and more exhaustive examinations.

A simple approach to this problem may be found in the classification of patients with gallbladder disease into three groups: (1) Those who have digestive disturbances but no colic, (2) those who have colic but no digestive symptoms, and (3) those who have both colic and digestive symptoms.

In studying patients of the first group, one must determine whether the digestive symptoms are in reality related to disease of the gallbladder or to some other disturbance. One may likewise be called upon to evaluate such vague manifestations as exhaustion, insomnia, dizziness, pains and aches in and about the muscles and joints, and headaches, all of which are common to toxic conditions of various origin. Again, the patient may describe symptoms resembling those of duodenal ulcer. If, as occasionally happens, a duodenal ulcer is also present, a proper classification may be practically impossible.

Especially is confusion likely to arise in the differentiation from appendicitis. Right iliac pain is not uncommon in cholecystitis and, on the other hand, a high lying inflammatory appendix may be mistaken for an infected gallbladder. This fact, and the fact that the two diseases are frequently associated calls for a fine interpretation of all the symptoms and signs and the use of all the diagnostic measures at our disposal.

Patients of the second group, i.e., those with upper abdominal colic but no digestive symptoms, should be studied not only for disease of the gallbladder, but for disturbed liver function, disease of the pancreas, a gastrointestinal infection or angina pectoris. If the gallbladder is the offender, stones will usually be found and a penetrating history will reveal that the patient has had a mild disturbance for several years, or perhaps an early period of severe symptoms followed by a period of comparative quiescence. Epigastric colic, however, is frequently associated with non-calculous cholecystitis, the causative agent being pericholecystitis, spasm of the gallbladder and ducts and of the sphincter of Oddi, or some type of mechanical obstruction outside the ducts. When the colic begins as a mild ache and is associated with a digestive disorder, one should immediately suspect cholecystitis.

Some light may be thrown on the various diagnostic problems of gallbladder disease by gastric analysis and duodenal drainage. The latter is most valuable as a means of studying the concentration and emptying function of the gallbladder. More important, however, is the roentgenographic study, without which no examination is

complete. The demonstration of a poorly functioning organ in a patient who presents symptomatic evidence of cholecystitis is ample indication for surgical interference. On the other hand, poor function alone may be of nervous or other origin and thus is not sufficient reason for operation.

Regardless of the roentgenographic picture, when the symptoms of cholecystitis are clear cut, severe and persistent, operation is advisable. The roentgenogram is not always a dependable means of determining the true pathologic condition; even in well established cases of cholecystitis, including those with stones, it occasionally fails to reveal either stones or a marked alteration of function.

There is no question as to the necessity for operation when stones are demonstrated. The vast majority of these patients give a history of colic and many of them report having had some degree of jaundice. Even though the symptoms may be mild and the function of the gallbladder appears little altered or not at all, it is the better part of wisdom to rid the patient of this source of trouble, as a precaution against an acute catastrophe. Another possibility to be borne in mind is that of malignancy. Carcinoma of the gallbladder and ducts is relatively rare, yet it is significant that stones are found in almost every case.

From pathologic studies made in an effort to determine which type of chronic cholecystitis is most amenable to relief by surgery, it appears that removal of the thick walled gallbladder with stones and associated cholesterosis (the "strawberry" gallbladder) practically always affords complete relief, provided the liver and other organs have not been too severely damaged. The next best results are obtained following cholecystectomy for simple cholecystitis with stones and, finally, following removal of the non-calculous gallbladder with extensive inflammatory reaction and cholesterosis or papillomas.

Once the indications are established, the earlier operation is performed the better. Cholecystitis is a recurrent and progressive disease, and many surgical deaths are in reality medical deaths, from lack of surgical treatment until the adjacent organs have become hopelessly involved in the pathologic process. Statistics show that the mortality increases commensurately with the duration of the gallbladder symptoms. Operations upon the common duct, frequently necessitated by long standing cholecystitis, further increase the hazard.

Acute Cholecystitis. One of the strongest arguments in favor of early operation in chronic cholecystitis lies in the danger of an acute attack and possible perforation. In many cases of acute cholecystitis, the attacks are preceded by a long standing chronic cholecystitis and are induced by obstruction of the cystic duct by a stone and a superimposed infection. In others, they may be provoked by strangulation and edema of the ducts from some other cause.

It appears, however, that obstruction of the duct and infection are not the only factors in the production of acute cholecystitis. There is considerable experimental evidence that it may be of chemical origin. In a personal communication, W. D. Gatch calls attention to the fact that cholecystitis is rare in children, though infections are more common in children than in adults, and, further, that abdominal wounds rarely become infected following operations for acute cholecystitis. He likewise points out that the bile is sterile in the majority of cases of cholecystitis. In addition, it has been shown that bile salts introduced into the gallbladders of animals will precipitate an acute attack, as will pancreatic juice. Clinically, the condition produced is identical with that of acute spontaneous cholecystitis in man.

In this theory may be found an explanation for the fact that many cases of acute cholecystitis subside under treatment. It also explains those cases in which no stone is found in the duct at operation, the bile is sterile on culture, and infection is conspicuously lacking. From my own experi-

ence, the theory is entirely tenable, and I believe we should broaden our view to include a chemical etiology as well as an obstructive and infectious origin for the acute process.

As to the management of acute cholecystitis, in the first place the patient should be placed in the hospital at the earliest possible moment after the beginning of the attack. He can thus be better assured of receiving the care suited to his particular needs and can be promised a more favorable prognosis. In the second place, measures should be undertaken immediately to restore the depleted fluid balance in the tissues and otherwise prepare the patient for operation. Aside from this, the management must rest upon the findings in the individual case. Here, again, the difficulty is that the symptoms do not always reflect the true pathologic condition.

The clinical signs which are regarded as demanding immediate surgery are (1) sustained abdominal pain with rigidity, (2) a tender mass in the right upper quadrant, (3) an elevation of temperature and (4) a rising leukocyte count. Especially are sustained pain and a tender mass positive indications. Early in the attack, a mass may not be palpable, rigidity and tenderness may not be pronounced, and the temperature, pulse and leukocyte count may be essentially normal. In these cases, operation may be delayed while preparatory treatment is given. If the patient is in good condition, preparation over a period of twelve to twenty-four hours is sufficient. Often, however, a more extensive preparation is desirable, especially for patients who present a poor risk. Fortunately for these, the attack will usually subside, permitting operation at a more advantageous time. By keeping a close watch of the patient and the use of proper laboratory studies, one can generally determine what course the acute process is going to take within twenty-four to thirty-six hours after the onset. If, during this period, the symptoms do not begin to subside or if they become aggravated, operation is urgent.

One cannot discuss the subject of cholecystitis without referring to the bile ducts, because of the frequent association of duct disease with cholecystitis and the damaging effect of their obstruction upon liver function. Much of the current discussion is centered about the indications for opening the common duct. As is well known, obstructions necessitating choledochotomy may arise from stricture, carcinoma of the duct or pancreas, pancreatitis or other abnormalities, yet the vast majority are produced by a stone or stones.

In the hands of experienced surgeons, the practice of openly exploring the ducts is increasing. One reason for this is the fact more patients are being operated for cholelithiasis and fewer for non-calculous cholecystitis. Another is that the duct may contain stones even though the gallbladder contains none, and still another, the possibility of reformation of stones in the duct subsequent to cholecystectomy alone. Although these are excellent reasons, the fact remains that the duct should be opened only in the presence of ample indications. The mortality of the operation with either cholecystectomy or cholecystostomy is high. Admittedly, complications incident to advanced disease are responsible for most of the deaths, yet the procedure is often technically difficult and, in any case, adds to the surgical trauma and thus further taxes the patient's recuperative powers.

The findings commonly accepted as justifying choledochotomy may be enumerated as follows:

1. *Palpation of a Stone.* This is not always an easy matter. Moreover, one should take care not to mistake an enlarged gland for a stone in the duct.

2. *Abnormal Dilatation of the Duct.* Again, one should be sure that the dilatation is not functional, in compensation for loss of gallbladder function, or that it is not due to extrinsic mechanical obstruction. Or, the dilatation may be secondary to gallbladder disease and amenable to relief by cholecystectomy alone. If there is

thickening of the duct walls and evidence of infection within, however, choledochotomy is advisable; even though a stone should not be found, drainage should be instituted.

3. *Contracted Gallbladder.* When one encounters a gallbladder no larger than the thumb, one may be sure of a long standing infection. The gallbladder may not contain stones, yet is more than likely that they will be found in the duct.

4. *Multiple Small Stones in the Gallbladder with an Enlarged, Patent Cystic Duct.* Stones too small to be detected by the palpating hand will frequently pass from the gallbladder into the common duct. This may happen during mere manipulation of the gallbladder. In such cases, it is not only advisable to open the duct, but the cholecystectomy should be performed first in order that one may be sure the duct is entirely free of stones at the conclusion of the operation.

5. *Jaundice or a History of Jaundice, Especially if Associated with Chills and Fever or with Gallstone Colic.* Since these symptoms may also be produced by disturbances other than a stone, a thorough search should be made for an obstructing lesion before the duct is opened. It should be borne in mind, moreover, that duct stones may be present without jaundice.

6. *Flocculent Bile in the Duct.* When the presence of a stone seems doubtful, a small quantity of the contents of the common duct should be aspirated. If the bile is cloudy and flocculent, the duct should be explored and drained. Not infrequently, an obstructing stone will be found at the lower end.

To preclude a second choledochotomy, every possible means should be employed to clear the ducts at the initial operation. The exploration should include the hepatic as well as the common duct. The ampulla, also, should be thoroughly searched with scoops for any possible stones hidden in its recesses. In addition, the patency of the distal end of the duct should be demonstrated with a probe; not until this has been done may one feel reasonably sure that the patient will have no further trouble. Should an obstruction be found at this point, the sphincter may be dilated almost to the size of the normal common duct without injury and with little fear of subsequent contraction, provided one uses bougies of graduated size. Finally, drainage should be instituted by means of a T-tube. The tube is brought out through a stab wound in the side and is not withdrawn until bile flows freely into the intestinal tract and there is no obstruction in the biliary tree, as determined by cholangiograms.

A vital influence in the success of operations for gallbladder and duct disease is the preoperative and postoperative treatment. Patients without evidence of long standing cholecystitis may not require any preparation other than the routine dietary restrictions and forced fluids. Those with advanced disease, however, should receive treatment directed especially toward conservation of liver and kidney function, as indicated by appropriate tests. The presence of jaundice increases the need for intensive preparatory measures. The prothrombin level in the blood should be determined and any hemorrhagic tendency counteracted, both before and after operation, by transfusions and the administration of vitamin K.

The icterus index affords an effective means of detecting and studying jaundice. Repeated tests will show whether the jaundice is deepening or subsiding. If possible, operation should be delayed so long as the index is rising or fluctuating, as the risk is less after the jaundice recedes to some extent.

When the common duct has been opened, liberal quantities of Decholin or some other cholagogue will be found advantageous in stimulating the flow of bile and promoting better drainage after operation. This treatment may be continued indefinitely.

Another important factor in the safety of the operation, and certainly the greatest aid to the surgeon is ample exposure of the

operative field. In the majority of cases, an upper right rectus incision, its middle over the region of the common duct, is suitable. In obese patients, however, the supraumbilical transverse incision is most advantageous, in that the tissues of these individuals are usually friable and this approach provides a safeguard against wound disruption and hernia.

When feasible, cholecystectomy is eminently preferable to drainage of the gallbladder. The mortality of the operation is low, recovery is rapid, and no further surgery is necessary. The risk of a primary cholecystectomy, moreover, is far less than that of a secondary procedure. If, however, the patient's condition is poor or if the inflammatory process is so extensive that identification of the ducts and vessels is impossible, it is better to insert a tube into the gallbladder and close the abdomen. One's chief concern should be the safety of the patient, and drainage may be a lifesaving measure, particularly in the presence of a perforation or following exploration of the ducts. The gallbladder may then be removed at a more advantageous time. Subsequent cholecystectomy is usually advisable, as a precaution against irreparable damage to the liver.

If the inflammatory process is severe, the technic of cholecystectomy may present some difficulties. Dissection should be carried out in a dry field, and one should be sufficiently familiar with the vessels and ducts to avoid their injury. The ducts and vessels are often abnormally situated, either naturally or because of edema or displacement by adhesions, and accessory ducts are commonly encountered. Incision or ligation of these anomalous or displaced structures may lead to serious consequences. No doubt, many so-called "liver deaths" are the result of damage to the blood vessels during removal of the gallbladder, and duct strictures incident to surgical trauma are found in a conspicuous number of operations upon the biliary tree.

The cystic duct should first be isolated, its relation to the common and hepatic ducts determined, and the cystic duct and artery ligated before proceeding with removal of the gallbladder. In chronic or elective cases, dissection is begun at the cystic duct and continued from below upward, whereas if the condition is acute and the gallbladder greatly distended, dissection is begun at the fundus and carried downward. When jaundice is present, however, exploration is first carried out to determine the cause of the obstruction. Should an acute pancreatitis or a malignancy be found, it may be necessary to conserve the gallbladder. In these cases, the gallbladder is usually not materially diseased and may be successfully utilized for anastomosis to the intestinal tract.

The necessity for drainage of the gallbladder in the presence of infection is well understood. In other cases, whether to drain or close the abdomen tight following cholecystectomy depends more or less upon the finding of accessory bile ducts and whether or not the operative field can be made perfectly dry. As a rule, it is well to insert simple Penrose drains, bringing them out through a stab wound to the side of the incision. The patient experiences little inconvenience, and the drains are generally removed after four or five days.

A recent review of 803 surgical cases of cholecystitis revealed that 528, or 65.8 per cent, were of the chronic type; 132, or 16.4 per cent, were subacute; and 143, or 17.8 per cent, were acute. These findings and the mortality of each group are shown in the following table.

TABLE 1
CLASSIFICATION OF 803 CASES OF CHOLECYSTITIS, WITH MORTALITY

	Cases		Mortality	
	No.	Per Cent	No.	Per Cent
Chronic	528	65.8	13	2.5
Subacute	132	16.4	6	4.5
Acute	143	17.8	14	10
Totals	803		33	4.1

Stones were found in 66 per cent of these fatal cases, and perforation had taken place in 40 per cent.

In the 803 cases, 682, or 85 per cent, of the operations were upon the gallbladder alone, while the ducts were opened and drained in 121, or 15 per cent. Ninety-seven of the choledochotomies were primary and twenty-four were secondary. (Table II.)

TABLE II
OPERATIONS IN 803 CASES OF BENIGN DISEASE OF THE GALLBLADDER AND DUCTS

		Mortality	
		No.	Per Cent
Primary gallbladder operations without choledochotomy	653	17	2.6
Secondary gallbladder operations without choledochotomy	29	2	7
Primary gallbladder operations with choledochotomy	97	9	9.3
Secondary gallbladder operations with choledochotomy	24	5	20
Totals	803	33	4.1

The nineteen fatalities following operations upon the gallbladder alone constitute approximately 2.8 per cent of the 682 in this group, whereas the fourteen fatalities following the 121 combined gallbladder and duct operations constitute 11.6 per cent. Thus, the mortality was more than four times higher in the latter group. This is probably due to the fact that stones are usually associated with long standing disease in which other structures have become involved.

It is of some significance, also, that in 228 operations performed within the past two and one-half years, we have found stones in the gallbladder in 82 per cent, and in forty-four duct operations (20 per cent of 228) have found stones in the duct in forty, or 90 per cent. Although the number of our choledochotomies has not materially increased within recent years, the recovery of duct stones has increased from sixty-six to 90 per cent. That stones were found in 90 per cent of only 20 per cent of choledochotomies indicates that the number in which the operation is performed is of far less importance than the use of sound judgment in observing the indications for the procedure.

CONCLUSIONS

Much of the success of gallbladder surgery depends upon the care with which patients are selected for operation. Aside from those who present both clinical and roentgenographic evidence of cholecystitis, there are a number of patients whose symptoms and roentgenographic findings are almost impossible of proper evaluation. Indeed, cholecystitis may mimic a wide variety of pathologic conditions. Probably the chief difficulty is encountered in the differential diagnosis of chronic non-calculous cholecystitis, as indicated by the fact that the larger number of poor surgical results are encountered in cases of this type. The desirability of exhaustive histories and meticulous studies cannot be too strongly emphasized. It is significant that the vast majority of patients who are completely relieved by operation are found to have gallbladder or duct stones, or both. Surgical opinion is almost universally in favor of removal of the gallbladder, when feasible, in the presence of stones.

Of material influence on the fate of these patients is early operation. When operation is delayed, the pathologic changes which take place throughout the biliary system not only further impair the patient's resistance, but necessitate more intensive preoperative and postoperative treatment, as well as more extensive surgery, and multiply the difficulties of the technical performance.

In acute cholecystitis, one is often confronted with the necessity for deciding between immediate and delayed operation. Usually, the attack will subside under appropriate treatment, permitting operation at a more advantageous time. One can

generally tell what course the acute process is going to take within twenty-four to thirty-six hours after the onset. A vital factor in the success of the treatment is the immediate hospitalization of the patient at the beginning of the attack.

Open drainage of the common duct is indicated when a stone is palpated in the duct, when the duct is abnormally dilated, when the gallbladder is contracted or contains multiple stones and the cystic duct is enlarged and patent, when the duct contains flocculent bile, and when the patient is jaundiced or gives a history of jaundice. By adherence to these criteria, stones will be recovered from the duct by choledochotomy in approximately 90 per cent of cases. The indiscriminate opening of the duct is not justified and, by the inexperienced especially, should be undertaken with extreme caution.

In addition to these considerations, no operation should be performed upon the gallbladder or ducts without adequate preparation of the patient. When necessary, this should include measures to conserve liver and kidney function, to control hemorrhage and to counteract infection. At operation, ample exposure of the field, with care to identify the important structures, and postoperatively, measures to correct any physiologic and organic disturbances will have a favorable effect upon the outcome.

MEDICAL treatment sometimes relieves symptoms, but never removes stones from the gall bladder. Unless there is some definite contraindication to operation the correct treatment for stones in the gall bladder is cholecystectomy.

From "A Short Practice of Surgery" by Hamilton Bailey and R. J. McNeill Love (H. K. Lewis & Co. Ltd.).

ACUTE PANCREATITIS*

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THE great diversity of opinion on the classification, etiology, symptomatology and treatment of acute pancreatitis attests to our limited knowledge of this disease. Although the condition is comparatively rare, it frequently presents a major surgical problem, carrying with it a high mortality. Careful analysis of clinical experiences and statistical studies of autopsy findings have given us added information of acute pancreatitis. In an effort to substantiate and increase this information, we have carefully analyzed the histories and pathologic findings of proven cases of acute pancreatitis occurring in the Veterans Administration Hospital at Hines, Illinois, during the past twelve years. The diagnosis was established in five patients at the operating table; three recovered and two died and had the diagnosis confirmed at necropsy. Fifteen additional patients were not operated upon but had the diagnosis established at autopsy. As there were 4,810 necropsies performed during this time, the seventeen deaths due to acute pancreatitis give an incidence of 3/10 per cent. Weiner and Tennant analyzed 4,000 autopsies performed at the New Haven Hospital and found thirty-eight cases of acute pancreatitis, an incidence approximately twice that of our series.

To avoid inclusion of information from possible diagnostic errors, we have analyzed only those cases in which the existence of acute pancreatitis was confirmed at operation or autopsy. Patients with a clinical diagnosis of acute pancreatitis who recovered without surgical intervention have not been included in this study. Patients in whom a questionable diagnosis of edematous pancreatitis was made at operation but in whom fat necrosis was not found have been eliminated from this series.

CLASSIFICATION

The classification of degrees of inflammation of the pancreas has varied markedly with different authors. Early classifications of acute, subacute and chronic inflammation were given. Fitz, in 1889, divided acute pancreatitis into hemorrhagic, gangrenous and suppurative. He described the hemorrhagic type as "peracute," and the gangrenous as acute, believing that it generally resulted from hemorrhagic pancreatitis. In more recent years authors have quite generally agreed on a classification of acute edematous pancreatitis which is a fairly mild form, and on a more fulminating and severe variety referred to as acute hemorrhagic pancreatitis or acute pancreatic necrosis. Boyd points out that in this latter form there may be little or no hemorrhage, and the lesion is more necrotic than inflammatory. Coffey believes the difference between these two types is the lack of intraglandular activation of trypsinogen in the edematous type. Cole suggests that the edematous type is the result of obstruction of the ducts, whereas the necrotic type is the result of tryptic digestion within the gland. In our series of seventeen autopsy reports, 65 per cent were found to have the acute hemorrhagic form of pancreatitis. The remaining 35 per cent were diagnosed as acute suppurative pancreatitis. Fat necrosis and bloody fluid within

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FIG. 1. Fat necrosis and tissue destruction of omentum removed at necropsy from a patient who died of acute pancreatitis.

the peritoneal cavity were present in all cases. Six of the patients had abscesses of capacities varying from 10 to 500 cc.

ETIOLOGY

The frequent association of biliary tract disease with acute pancreatitis strongly suggests a close correlation between these diseases. Morton reports that 62 per cent of a series of ninety-seven cases had antecedent biliary tract disease. Lampson found that 55 per cent of twenty-nine patients with acute pancreatitis had gallstones. de Takats and MacKenzie noted an involvement of the biliary tract in 81 per cent of twenty-two cases, and Connell found gallstones in twenty-four of twenty-six patients with acute pancreatitis with fat necrosis. Carter and Hotz reported stones in the common duct in seventy-eight of their series of acute pancreatitis, while Cole found cholecystic disease in all eight of his patients seven with stones in the gallbladder and five with stones in the common duct as well. In our series of twenty cases, 55 per cent were found to have gallbladder disease. Cholelithiasis was found in eighteen and two of these also had stones in the common duct. Multiple liver abscesses also were found in two patients. In contrast to these reports, Weiner and Tennant found a co-existence of biliary tract disease in only 16 per cent of their necropsy findings of acute pancreatitis.

A number of theories have been advanced as to the mechanism by which biliary tract disease initiates acute pancreatitis. The transmission of bile into the pancreatic ducts has been advanced as an etiologic factor. Having found a stone impacted in the ampulla of Vater which permitted bile to be short circuited into the pancreatic duct, Opie advanced the theory that blockage of the ampulla, converting the common duct and the duct of Wirsung into a single channel, permitted bile to enter the pancreas and was the etiologic factor of pancreatitis. Archibald believed this same mechanism could result from spasm of the sphincter of Oddi. Robins believes that in the necrotic type, bile initiates the original necrosis. Several known facts argue against the theory that bile entering the pancreatic ducts produces acute pancreatitis. McCaughan found that this disease could be produced in dogs by

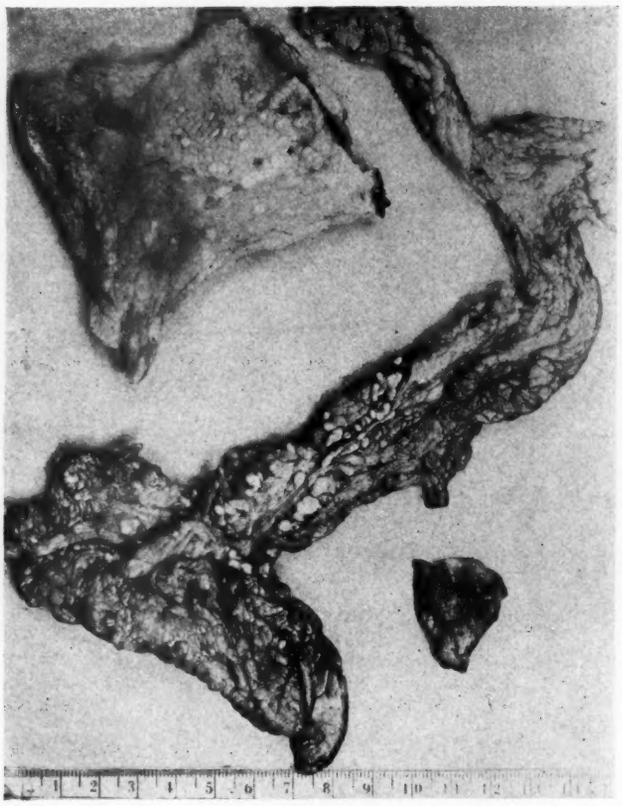

FIG. 2. A, the external appearance of the pancreas showing extensive necrosis.

the injection of bile, gastric juice or virulent bacteria only if sufficient pressure is used to rupture the pancreatic ducts. The necessary pressure is higher than that known to occur in the choledochus under any conditions. Anatomic studies have shown that only rarely is it possible for bile to enter the pancreatic ducts from the choledochus in man. Necropsy studies on patients with acute pancreatitis have generally proven that it was impossible for bile to enter the pancreatic ducts from the common duct. In our series of autopsies the relationship of the common bile duct to the pancreatic duct was accurately described in four cases. In one of these there was a common ampulla. In another, the openings were separate but adjacent at the papilla of Vater. In the third, the pancreatic duct inserted into the common duct. In the fourth, the pancreatic duct entered the duodenum 2 cm. lateral and proximal to the papilla of Vater. The work of Wolfer, who found that the secretory pressure of the pancreas is greater than that of the liver, further argues against the bile theory in the etiology of acute pancreatitis.

The transmission of bacteria from a diseased biliary tract to the pancreas has been considered as an etiologic factor in acute pancreatitis. Theories have been advanced that this infection could be transmitted by the blood stream, by the lymphatics, by direct extension or by transmission through the ducts. Robins, Dragstedt et al. agree that although frequently organisms can be cultured from the normal pancreas, the overwhelming toxicity in acute pancreatic necrosis is due to the results of protein decomposition rather than to toxins produced by bacteria.

Obstruction of the pancreatic duct may be an important etiologic factor in the production of pancreatitis. The frequency of the association of biliary tract disease with inflammation of the pancreas strongly suggests this. Stones in the common duct may cause pressure on the duct of Wirsung.

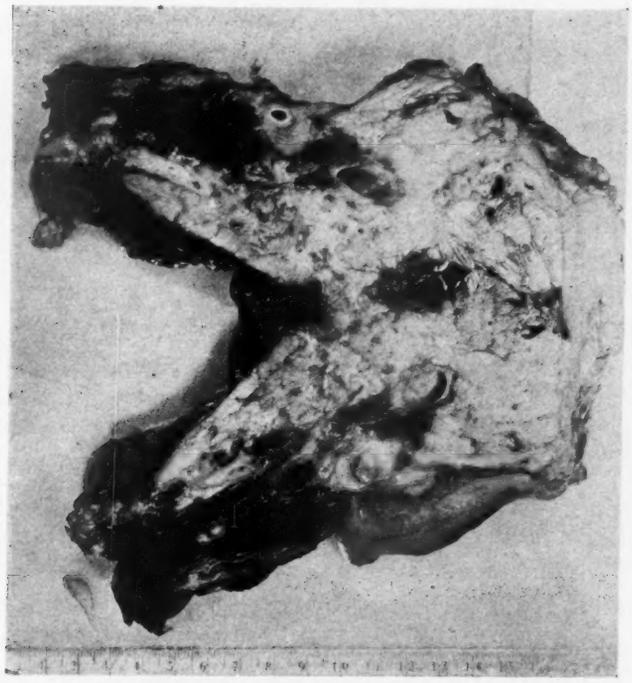

FIG. 2. B, the pancreas sections showing extensive hemorrhage and necrosis.

Swelling and edema of the extrahepatic biliary tract and its surrounding structures may do likewise. Rich has suggested that metaplasia of intraductal cells within the pancreas also might produce obstruction. In our series of seventeen necropsies for acute pancreatitis, definite obstruction of the pancreatic duct was found in five cases. In two, the ducts were blocked by fibrous tissue; in one, by a duodenal ulcer adjacent to the duct; in one, by an adenocarcinoma of the ampulla of Vater; and in the fifth, by a suture which had been placed about the pancreatic duct during the course of a gastrectomy performed for carcinoma of the stomach four days prior to the onset of symptoms of pancreatitis. In another case, acute pancreatitis and death occurred five days after a partial gastrectomy for a penetrating duodenal ulcer which had been dissected from the head of the pancreas.

Certain dietary habits are believed to be factors in the development of acute pancreatitis. Many attacks appear to follow the ingestion of large meals. Weiner and Tennant made the interesting observation that in fifty-one deaths following the excessive ingestion of alcoholic beverages, acute pancreatitis was found in twenty-five cases. Rhodes mentions external trauma as the direct etiological agent in five of his series of thirty-five patients with acute pancreatitis.

SYMPTOMATOLOGY

The symptoms of acute edematous pancreatitis are similar to those of acute hemorrhagic pancreatitis but of a milder degree. Pain is almost universally present, varying in degree with the severity of the pancreatic involvement. Its onset was sudden in 60 per cent of our cases and it occurred in the epigastrium in 70 per cent. It was diffuse in two cases, predominantly in the right upper quadrant in three, in the lower abdomen in two, and in the right lower quadrant in two patients. It was more pronounced on the left in only one patient. All patients with pain in the right upper quadrant had associated gallbladder disease. Although mention is often made of radiation of pain to the back, this occurred in only one of our patients. In approximately half of our patients the pain was sharp and severe, and in the remainder it was described as dull. Nausea and vomit-

ing universally was present. Three patients manifested chills and all subsequently were found to have pancreatic abscesses. Prostration or shock was found in over half of this series. None of the patients who survived surgery were in shock before operation. The admission temperature of these patients varied from 97° to 104°F., averaging 101°F. It is of interest to note that the preoperative temperature was normal in all patients who survived surgery. A history of previous attacks similar in character but milder in severity was given by four of these patients. Although eleven of the twenty patients were found to have disease of the gallbladder, only two gave a history of previous biliary attacks.

Examination revealed tenderness in all but four patients and these were either under the influence of opiates at the time of admission to the hospital or were moribund. The location of the tenderness corresponded roughly to the location of the pain. It was chiefly in the epigastrium in 50 per cent and chiefly in the right upper quadrant in another 50 per cent. This latter group all proved to have concomitant gallbladder disease. Left upper quadrant tenderness was marked in only one case. Muscle spasm corresponding to the area of tenderness was noted in 60 per cent of the patients. It is of interest to note that distention was mentioned as a prominent physical finding in all twenty of these patients. Audible peristalsis was more frequently absent than present. Jaundice has been frequently mentioned as a symptom of acute pancreatitis but was infrequent in our series. Many of the more serious patients manifested symptoms of shock consisting of a rapid pulse and low blood pressure with the characteristic restlessness and cold, clammy skin. Leucocytosis was invariably present in our series; the leucocyte count ranging from 11,000 to 45,000 and averaging over 20,000. All patients who recovered had a leucocyte count below 20,000. Glycosuria is an infrequent finding, being noted in six of thirty-seven patients by Elman, and in four of thirty cases by deTakats and MacKenzie. Metheny reports the presence of albuminuria in all of his thirty-two cases. Others have corroborated this observation and believe that the absence of albuminuria should make one doubt the existence of acute pancreatitis.

DIAGNOSIS

Before the use of serum amylase determinations as a diagnostic aid, the incidence of a correct diagnosis of pancreatitis prior to operation or necropsy was infrequent. It is believed that the use of this test will greatly improve diagnostic accuracy. In a series of sixty-four cases analyzed by McWhorter, a correct diagnosis was made in only 11 per cent. In a series of thirty-five cases analyzed by Lewison, a similar percentage of accuracy in diagnosis was made with pancreatitis being considered as an additional possibility in 6 per cent of the remaining patients. Elman collected a group of thirty-seven patients having acute edematous pancreatitis in which the correct preoperative diagnosis was not made in a single instance. He also made the interesting observation that twenty-four patients of this series had histories of previous attacks. deTakats and MacKenzie likewise reported that in their thirty cases a correct diagnosis had not been made in a single instance.

The determination of serum amylase is believed to be a valuable aid in the diagnosis of acute pancreatitis and most observers agree that the differentiation between edematous and necrotic pancreatitis often can be made by its use. Lewison emphasizes the importance of the amylase test, especially if the patient is to be treated conservatively. He found that the serum amylase values are constant in the human being and are not influenced by age, sex, diet, vitamin deficiency or starvation. He also observed that diseases of the liver and biliary tract depress the serum amylase whereas acute pancreatitis elevates it. Metheny et al. concluded from their study of thirty-two cases that in the necrotic

type of pancreatitis the amylase level returns to normal or below in two to three days but in the edematous type it requires an average of eighteen days for a normal value to be re-established. Morton agrees with these principles and believes that the serum amylase level is of the greatest importance in deciding whether or not to operate. Elman disagrees with these concepts and although he admits that the serum amylase tests will indicate a serious pathological condition in the pancreas, he does not believe that the two types of pancreatitis can be differentiated in this manner. Clasen et al. explain the fall of serum amylase to normal or below after the initial rise by postulating that it may be due to the "walling off" and fibrosis of the involved areas of the pancreas.

The preceding paragraphs enumerate the symptoms and findings which should suggest the diagnosis of acute pancreatitis. The patient usually gives a history of an acute onset of abdominal pain which may vary considerably in intensity. This pain is most frequent in the epigastrium and the right upper quadrant and rarely radiates to the back. There is associated tenderness and often rigidity in the area of pain. Nausea and vomiting invariably are present. If the disease is severe, the patient will present a picture of shock with a low blood pressure, rapid pulse, apprehension and a cold, clammy skin. There likely will be a history of previous milder attacks and often a history suggestive of biliary tract disease. Laboratory examinations will reveal a leucocytosis and probably an albuminuria. A serum amylase determination will show a definite elevation of the serum amylase if the test is made within two or three days of the onset of the symptoms.

DIFFERENTIAL DIAGNOSIS

With the present tendency toward conservative treatment of acute pancreatitis, it is most important to reach a correct diagnosis. Acute cholecystitis may be confused with acute pancreatitis and may exist in conjunction with the latter disease. Here the serum amylase test is of considerable value for if acute pancreatitis does not exist the serum amylase level should not be elevated. The most important disease to differentiate from acute pancreatitis is a perforated peptic ulcer. As this condition demands immediate surgery whereas pancreatitis is treated conservatively, every effort should be made to differentiate between these two abdominal emergencies. They often will present very similar pictures, especially if the pancreatitis is of the hemorrhagic type. There are certain diagnostic criteria which are of value in making a differential diagnosis. If a perforated peptic ulcer drains freely into the peritoneal cavity, it will produce a spreading peritonitis with a diffusion of pain gradually producing a generalized board-like rigidity of the abdomen. An *x*-ray plate taken with the patient in a vertical position usually will show air under the diaphragm. The serum amylase will not be elevated. Although many authors consider perforated peptic ulcers as silent ulcers, a carefully taken history frequently will elicit pre-existing ulcer symptoms.

Other acute abdominal conditions occasionally may simulate acute pancreatitis or be confused with it. An acutely inflamed or gangrenous appendix lying high behind the ascending colon may present a difficult diagnostic problem. An acute diverticulitis, especially with perforation, also may produce symptoms difficult to differentiate from those of acute pancreatitis.

TREATMENT

Surgical intervention which formerly was advocated for all cases of suspected acute pancreatitis has produced such poor results that more conservative treatment is generally replacing it. This is true especially of the acute edematous type of pancreatitis in which nonoperative therapy has been followed by a far greater incidence of recovery than any form of surgical intervention. The primary indication for early operation in acute pancreatitis is a questionable diagnosis. If a perforated viscus is

suspected and the patient is not in shock, an immediate operation may be indicated. Suspicion of an acute cholecystitis does not justify early operation because the danger of perforation of an acute gallbladder is much less than the increased mortality resulting from early surgery in a cholecystic disease. We believe this applies even where there is elevation of serum amylase levels.

We would like to outline the treatment of acute abdominal catastrophes which could be produced by acute pancreatitis. If a patient is admitted to the hospital in shock or in an extremely critical condition, immediate surgery usually will prove fatal. He should be put at rest and his pain relieved by morphine. His stomach should be kept at rest by avoiding all oral feedings and by continuous gastric suction. Whole blood and glucose solutions should be given intravenously to combat shock and to maintain fluid balance. Oxygen should be administered if indicated. Careful observation and *x*-ray studies should be utilized to determine the possible existence of a ruptured viscus. If this is suspected and the patient's condition permits, early surgical intervention is mandatory. If a ruptured ulcer is found at operation, it must be closed and adequately protected. However, if acute pancreatitis is found, any conditions bearing upon its etiology may have to be treated. If the biliary tract is diseased it should be decompressed. In most cases the acute nature of the disease and the critical condition of the patient will justify only the minimal amount of surgery to accomplish the release of biliary pressure. Cholecystostomy usually is the operation of choice. In this early stage, abscess formation in the region of the pancreas is rare and attempted drainage of an edematous or hemorrhagic pancreas is both impossible and useless.

If immediate surgery is not decided upon, intensive conservative therapy should be continued. Absolute mental and physical rest must be maintained as well as freedom from pain. Parenteral feeding of glucose, saline, proteins and blood should be continued and guided by careful laboratory studies. These should include estimations of blood chlorides, total proteins, careful and repeated checks on the red and white blood cell counts and an accurate check of fluid intake and output. Serum amylase levels should be followed closely to determine the presence of pancreatic involvement. Blood sugar levels will serve as a guide to the administration of insulin.

Subsequent care will depend largely upon the response of the patient. If clinical studies and serum amylase determinations suggest an acute pancreatitis secondary to a biliary tract disease, operative intervention should be urged when the patient's symptoms have subsided. It is well to wait for several weeks when the patient will be in better condition to withstand surgery so that the gallbladder may be removed and the common duct drained if the operative findings justify these procedures. A cholecystostomy almost invariably must be followed by subsequent cholecystectomy and it is well to delay surgery until adequate biliary treatment can be instituted at the first operation. If symptoms of a pancreatic abscess develop, this should be subsequently drained. It will be indicated by persistence of fever, of leucocytosis and frequently of a tender mid-abdominal mass. Such symptoms should suggest this even though the serum amylase level has returned to normal or below.

SUMMARY

The difficulty in differentiating between acute pancreatitis and other abdominal catastrophes is proven by the high incidence of diagnostic errors. It is most important to recognize perforations of peptic ulcers as immediate surgery is mandatory to good results. On the other hand, conservative treatment of acute pancreatitis gives a far lower mortality than that following early operative intervention. The determination of serum amylase levels has been a valuable addition to our diagnostic armamentarium as these rise sharply in

the early stages of acute pancreatitis but are not increased in other acute abdominal conditions.

The usual symptoms of acute pancreatitis are as follows: The history of acute abdominal pain varying in intensity and being most frequently located in the epigastrium and the right upper quadrant; tenderness and often rigidity in the area of the pain; nausea and vomiting invariably existing with shock accompanying severe attacks. There is often a history of previous milder attacks or of biliary colic. Jaundice is not infrequent. The leucocyte count is markedly elevated. Glycosuria and albuminuria frequently are found.

Conservative therapy consists of relief of pain, rest in bed, continuous gastric rest and suction, adequate parenteral feedings to maintain blood chemistry and fluid balance, blood transfusions to overcome shock and careful observation. When the acute symptoms have subsided and the patient's condition has improved, biliary tract surgery should be contemplated if evidences of disease are found. Late drainage of pancreatic abscesses may be indicated.

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TRAUMATIC LESIONS OF THE ABDOMEN WITH RUPTURED VISCERA

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ABDOMINAL injuries are usually serious. They may be divided roughly into penetrating and non-penetrating wounds, and intra-abdominal trauma without visible wounds of the abdominal wall. All penetrating and non-penetrating wounds require surgical exploration and débridement. All penetrating wounds also require careful systematic inspection of all viscera and the performance of the least surgical procedure compatible with saving life. Patients with intra-abdominal trauma without visible wounds of the abdominal wall often require a period of observation for diagnosis and may or may not require operation.

Successful treatment in the surgery of trauma depends in no small measure on the speed and efficiency with which the casualty is admitted to the hospital. If the mortality rate of injuries to the abdomen is to be maintained at a minimum, it is of prime importance that the time lag or the period between the incidence of the trauma and the consummation of definitive treatment be reduced to as short a time as possible. This means that the speed of transport of the casualty to a hospital or station where careful examination can be made and treatment be directed is of utmost importance. There is little that the layman can do at the site of injury except call for an ambulance with dispatch and inform the driver of getting the patient to the hospital as soon as possible.

The mechanism of trauma to the abdomen may be of many different types. The most common causes are as follows: (1) Direct blow to the abdomen by some blunt object such as a piece of wood or a tool that the workman is using; (2) a fall from a height of a few feet or more, the impact of landing producing the injury; (3) injuries resulting from stab or gunshot wounds, and (4) squeezing injuries, such as are caused by being caught between moving objects.

ANATOMY

In considering trauma to the abdomen the surgeon must keep in mind the anatomical structures which constitute the abdominal region. This region consists of the abdominal wall which is supported by the ribs and the spine and is anchored to a bony frame work, the pelvis, at the lower portion. The abdomen contains two types of organs, the solid ones, such as the liver, spleen, kidneys and pancreas, and the hollow ones, the stomach, the intestines, the gallbladder and the urinary bladder. The supports of the abdominal organs are more or less elastic and contain blood vessels which may be so injured that fatal hemorrhage follows. The solid organs have a more or less constant size and shape that undergo but little variation with changes in the physiologic status of the individual. The spleen becomes engorged and full after the ingestion of a meal, while the other solid organs vary but little, if any.

The hollow organs vary their size and shape considerably during the normal daily activities of the organism. After a meal the stomach will increase greatly in size as will the small bowel when food enters its lumen. The urinary bladder will vary greatly in size and become overstretched under tension. When an organ is distended it is far more liable to injury than when it is empty; consequently, abdominal injuries are more likely to produce severe damage

to the hollow viscera when they are sustained soon after the ingestion of food.

Shock and hemorrhage commonly accompany all three types of abdominal trauma and may be adequately treated by various measures. Hemorrhage is synonymous with shock in intra-abdominal trauma, and therefore, operation to control it should not be delayed.

When visceral injury is present the following signs* may be noted:

"1. Marked muscle resistance is one of the most reliable signs of visceral injury. It is usually quite marked, and if progressive is almost pathognomonic.

"2. Tenderness is often exquisite. In the early stages, it is localized in the region of injury; later it becomes more diffuse due to peritoneal irritation by infection or blood.

"3. Shock of variable degree; pallor, cold extremities, subnormal temperature, blood pressure and rapid pulse are almost invariably present. A progressive increase of pulse rate and fall in blood pressure with or without thirst, pallor and restlessness indicates continued hemorrhage. The so-called delayed or secondary shock is usually due to hemorrhage and should be considered.

"4. Dullness in the flanks, shifting on changing position indicates fluid, usually blood, but may be gastrointestinal content or urine and peritoneal effusion.

"5. Obliteration of liver dullness indicates pneumoperitoneum (free gas) and is due to perforation of the gastrointestinal tract. After several hours it may be due to gas elaborated by the bacteria causing the peritonitis.

"6. Blood in the vomitus or stool suggests gastrointestinal injury; blood in the urine indicates injury to the urinary tract.

"7. Peristalsis will usually be absent when the abdomen is auscultated. In the presence of intraperitoneal inflammation, there is reflex atony of the bowel in the effort to avoid movement and dissemination of the irritating material until localization can be completed by adherence about the area. The presence of food or laxatives, however, tends to produce peristalsis in spite of the normal quieting influence.

"8. The hemoglobin and red count usually do not show definite decrease in the first hour or two even though there is considerable hemorrhage because all parts of the blood are lost in equal proportions. However, the blood loss is rapidly rebuilt in total volume by absorption of fluid from the tissues, the gastrointestinal tract and from the intravenous administration of fluid at such times. This will produce a dilution of the remaining blood and the disproportion will show in decreased hemoglobin and red blood cells when a count is repeated in 2 or 3 hours. On the contrary, the white blood cells usually show an early marked increase both in the total count, in the percentage of polymorphonuclears and in proportion of immature cells. Hemorrhage may produce an excessive response of this sort to 30,000 or even above."

* The Cyclopedia of Medicine, Surgery and Specialties; Volume 1—page 25, W. Emory Burnett, M.D.

MANAGEMENT

When a patient has received an injury to the trunk, as often as not, there is little to guide the surgeon in the matter of external bruising. The problem confronting the surgeon is to decide and decide as quickly as possible whether or not the patient has sustained an intraperitoneal or intra-abdominal lesion. This is often a difficult matter, particularly since the patient with trauma to the abdomen is suffering from shock when first seen.

The prelude to the examination of an injury to the abdomen is to inquire of the patient, if he is able to comprehend and respond, as to the mechanism of the injury. Has he vomited since the accident? Did the vomitus contain blood; if so, it is more definite evidence of gastric injury. Has the patient passed urine since the accident, and was there any blood in the urine? Has there been any passage of blood from the rectum? Of what does the patient complain? Ask the patient to point with one finger where the pain is most acute and mark this location. Often the patient

may accurately locate the site of a perforation. Pain is important if localized and persistent.

Note and record the patient's temperature, the rate of the pulse, its quality and volume, respirations, and the blood pressure. Check and record the pulse, its quality, and blood pressure every fifteen minutes. A blood count including red cell count and white cell count, and estimation of hemoglobin should be made immediately, recorded and repeated every hour.

If the patient is able to urinate, a urinalysis should be made particularly checking for the presence of blood. If the patient is unable to urinate, catheterization should be resorted to with the caution not to use force and add more trauma if a ruptured urethra is suspected.

Roentgenograms showing the entire abdomen, including the pelvis and lower chest should be taken. In some cases it is well to take lateral views to look for areas of free gas in the abdomen.

On physical examination careful inspection should be made to notice any external evidence of injury such as swelling, abrasions, areas of redness or discoloration of bruising to determine if any fractures are present about the pelvis, spine or ribs; by palpation of the abdomen to determine if there are any local points of tenderness or muscular resistance. Areas of dullness or tympany should be noted and such areas marked with a pencil for checking at subsequent examinations.

The shock, when present, should be promptly treated. Body heat should be conserved; the foot of the bed may be elevated 12 to 15 inches; 500 cc. to 1,000 cc. of plasma when available should be given intravenously; and the judicious use of 500 to 1000 cc. of 5 per cent glucose in normal saline considered.

If after the above the extent of intra-abdominal injury is not evident, the patient should be put to bed for observation. Repeated examinations should be made at least every thirty minutes until a positive diagnosis or decision is reached. Do not give anything by mouth. While we appreciate the value of the relief of pain in combating shock, it is advisable to avoid sedative drugs, such as morphine, during the observation period as they tend to mask important symptoms.

The surgeon should make a positive diagnosis of intra-abdominal injury within less than six hours from the time of the accident, preferably within two to four hours if possible. A patient operated on within six hours, as a rule, has a good chance for recovery; after six hours the chance of recovery greatly diminishes.

In perforating wounds of the abdomen, such as gunshot or stab wounds, it is apparent that the abdominal wall has been pierced so that immediate operation to repair the defects should be performed. This type of injury is usually evident and need cause no delay in making a diagnosis and deciding the treatment.

In selecting an anesthetic, preference is given to nitrous oxide, cyclopropane or ethylene preceded by one or two injections of morphine-scopolamine, depending on the available time before operation is begun. We have found the use of curare (intocostrin) intravenously just preceding and during the inhalation anesthesia of material aid in providing desirable relaxation.

Squeezing injuries to the abdomen may result in injury to the hollow organs, such as the gastrointestinal tract or urinary bladder; may rupture blood vessels causing dangerous hemorrhage, and in addition may produce fractures or lacerations of the solid structures such as the liver, spleen, kidneys or pancreas.

Injuries to the liver and spleen may be treated conservatively if one can be reasonably sure that the patient is not developing a serious hemorrhage. With evidence of internal hemorrhage it is essential to open the abdomen and control the source of the bleeding. In addition to the usual signs of hemorrhage one should look for referred pain to the left shoulder (Kehr's sign) which is frequently present when blood is in contact with the undersurface of the diaphragm. Another valuable maneuver is the application of Saegesser's sign, apply-

ing deep finger-point pressure between the sternomastoid and the scalenus medius on the left side. If positive in the case of a ruptured spleen, even in cases of intracapsular hemorrhage the pressure elicits violent pain.

A ruptured spleen may be safely removed with no subsequent ill after-effects. We have never tried to suture the spleen but have removed it when necessary.

Injuries to the liver can best be managed by resorting to suturing or using a simple gauze pack or using omentum. The use of fibrin foam and thrombin or gelatine sponge have also been recommended to control bleeding effectively from small lacerations of the liver, spleen and kidneys.

Lacerations of the stomach and intestine should be closed; in the case of the intestines the openings are closed in such a manner that the completed suture line is transverse, thus increasing the diameter of the lumen. If the intestine is badly damaged or deprived of blood supply by mesenteric damage, resection and end-to-side or side-to-side anastomosis may be done. In the case of a rupture of a hollow viscus with peritoneal contamination with spillage of gastrointestinal content, the foreign material is removed by suction followed by a liberal use of sulfathiazole and suitable drainage.

In the postoperative care one may stress the use of penicillin intramuscularly or it may be added to whatever fluids are administered intravenously to maintain a proper fluid balance. Mention is also made of the use of the Wangensteen constant suction apparatus and the Miller-Abbott tube to relieve and combat gastric and intestinal distention.

Injuries to the kidneys are fairly common complications of abdominal trauma and are best treated in a conservative way. Hematuria, the most prominent symptom in renal injuries, is usually present from the onset and may persist for one to two weeks. Associated with the hematuria is pain in the back which increases in severity so as to require sedatives. There is also a moderate degree of shock present in most cases. A careful check on the blood pressure at intervals of one to two hours, as a rule, will give an indication as to the extent and severity of the injury. It is also important to keep a close observation on the blood pressure. If the patient's general condition goes along in a satisfactory manner, conservative and supportive management is indicated, although the use of drainage in the loin may be necessary later. If the patient's condition appears critical, it may be necessary to explore the kidney area so as to control hemorrhage and institute drainage. In some cases it is necessary to do a prompt nephrectomy or resort to a pack.

A rupture of the bladder can be anticipated in injuries about the pelvis, especially if fracture is present. The urine should be examined for presence of blood. Additional investigation should be made by injecting a known amount of solution into the bladder and by being able to withdraw a like amount. If evidence of rupture is found, prompt repair is necessary.

SUMMARY

The following points should be observed:

1. Prompt transportation of the injured to a hospital or station where definite treatment may be executed.
2. Careful history as to mechanism and severity of injury.
3. Examination of the injured with making of records and prompt laboratory procedures. Repeated examinations at frequent intervals until a surgical opinion has been reached.
4. Prompt use of measures to combat shock and control hemorrhage; operation when indicated within the first six hours if possible. It is better to operate in cases which present indefinite and obscure findings with a possibility of serious abdominal injury, than to consider conservative observation too long.
5. Chemotherapy with the use of sulfa drugs and penicillin to combat infection.
6. Judicious use of methods to maintain a proper fluid balance and to combat distention postoperatively.

PROGRESS IN THE TREATMENT OF ACUTE APPENDICITIS*

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IN the past two decades four major contributions to the field of surgery were applied to the treatment of acute appendicitis. These were: (1) intragastric suction, (2) intravenous fluids, (3) whole blood and plasma transfusions, and (4) chemo and antibiotic therapy. The fact that these improvements came consecutively rather than concurrently permits us to evaluate their effects upon the treatment of appendicitis, together with other principles less influenced by time. For this purpose we have selected three representative periods in the history of the Cook County Hospital: (1) patients treated between 1928 and 1932 to whom intravenous fluids and intragastric suction were available; (2) patients treated during 1937 and 1938 when the use of blood was made available by the establishment of a blood bank; and (3) patients treated during 1944 and 1945 who were given sulfonamide and/or penicillin in addition to other adjuvant therapy. The first two groups of patients were first reported by one of us (K. A. M.) in an earlier report[1] but they will be utilized as a basis of comparison for more recent methods of treatment.

STATISTICAL SURVEY

This review includes an analysis of 5,543 patients with acute appendicitis treated at the Cook County Hospital. The care of these patients is largely entrusted to surgical interns and residents. The patients come from an indigent population who resort to self-medication and treatment for many hours or days before reporting to a hospital. The results, therefore, can never equal those reported by private institutions. Furthermore, war-time prosperity has caused a marked decline in the number of patients.

TABLE 1.
MORTALITY RATE OF 5,543 CASES OF ACUTE APPENDICITIS.

Year	Acute Appendicitis						Total Number Cases	Overall Mortality Rate, Per Cent
	Cases Without Perforation	Mortality Rate, Per Cent	Cases With Perforation	Mortality Rate, Per Cent	Cases With Abscess	Mortality Rate, Per Cent		
1928 to 1932	1,857	1.1	519	26.4	481	12.2	2,857	7.6
1937 and 1938	1,585	0.39	262	23.3	249	6.8	2,094	3.9
1944 and 1945	281	0.78	136	13.9	75	5.3	592	4.3

MORTALITY RATE. In Table 1 this material is divided according to a classification which we consider important, both from the point of treatment and prognosis: acute appendicitis *without* perforation, acute appendicitis *with* perforation, and acute appendicitis with abscess. Between 1928 and 1932, 1,857 patients with acute appendicitis without perforation were treated by appendectomy with a mortality of 1.1 per cent. During 1937 and 1938, 1,585 such patients were treated with a mortality rate of 0.39 per cent. During 1944 and 1945, the number of such pa-

* From the Hektoen Institute for Medical Research of the Cook County Hospital, the Department of Surgery of the Cook County Hospital and the Cook County Graduate School of Medicine, and the Department of Surgery, Northwestern University Medical School.

tients was 281 and the mortality rate 0.78 per cent.

During the period from 1928 to 1932, 519 patients with acute appendicitis with perforation were admitted and the mortality rate in this group averaged 26.4 per cent. During 1937 and 1938, 262 such cases were admitted and the mortality rate was 23.3 per cent. In 1944 and 1945, 136 patients with acute perforative appendicitis were admitted with a mortality rate of 13.9 per cent.

In 481 patients with acute appendiceal abscess during the years 1938 and 1932, the mortality rate was 12.2 per cent. In 249 such patients admitted in 1937 and 1938, the mortality rate was 6.8 per cent. For seventy-five such patients admitted during 1944 and 1945, the mortality rate was 5.3 per cent.

Comparing the over-all mortality rate for all three types of cases treated during each period, we found that in 2,857 patients treated during the 1928 and 1932 period, the mortality rate was 7.6 per cent; for 2,094 patients treated between 1937 and 1938, the mortality rate was 3.9 per cent; of 592 patients of all three types of appendicitis treated during 1944 and 1945, the mortality rate was 4.3 per cent. The latter figure requires interpretation because the census of patients with acute nonperforative appendicitis showed a far greater decrease than did the other two more serious types of appendicitis.

Influence of Surgical Intervention in Patients with an Acute Appendiceal Abscess. Table II represents a more detailed analysis of cases of acute appendiceal abscess treated with and without operation. There were 346 such patients treated by removal of the appendix and/or drainage of the abscess during the two periods, 1928–1932 and 1937–1938, with a resultant mortality rate of 18 per cent. During the same periods 284 patients were treated conservatively (Ochsner regimen) and the mortality rate was 4.2 per cent.

During the years 1944–1945, twelve patients with an acute appendiceal abscess were operated upon with a mortality rate of 8.5 per cent; in sixty-three patients who were not operated upon the mortality rate was 3.1 per cent.

TABLE II
INFLUENCE OF SURGERY UPON MORTALITY RATE OF PATIENTS WITH AN ACUTE APPENDICEAL ABSCESS

Year	Operated		Non-operated	
	Number of Cases	Mortality Per Cent	Number of Cases	Mortality Per Cent
1928–32 and 1937–38	346	18	284	4.2
1944–45	12	8.5	63	3.1

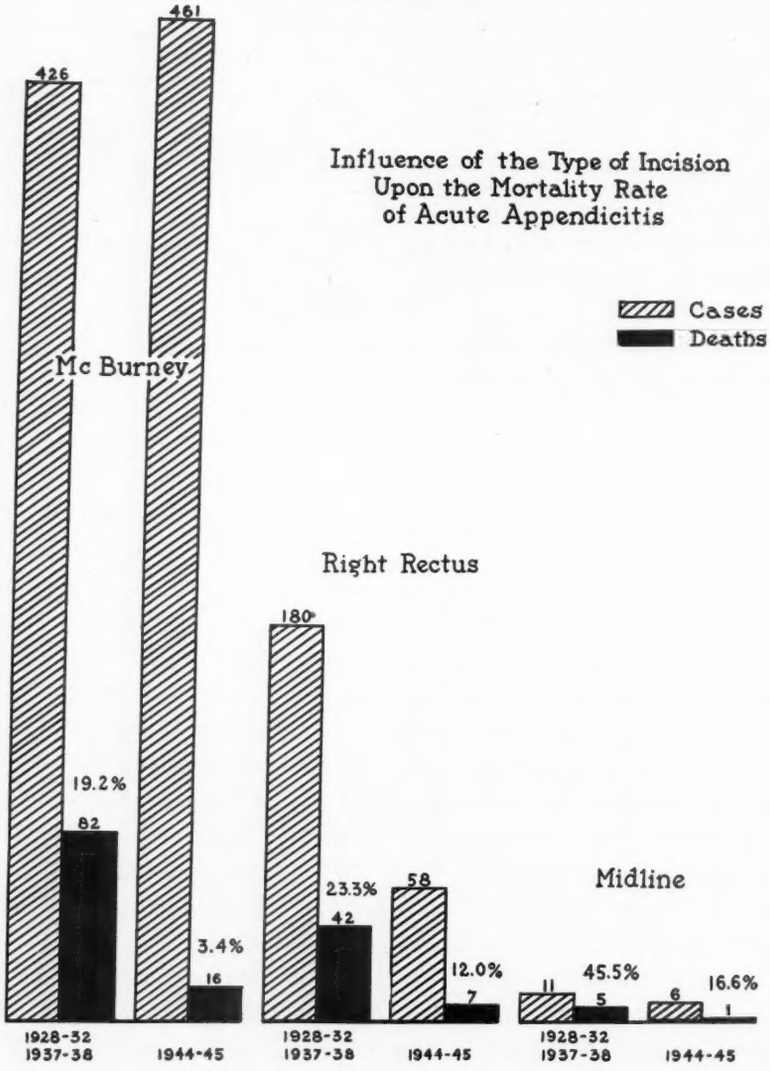
Influence of Incision upon Mortality Rate. The incisions employed for appendectomy in these patients were usually of three general types with slight modifications: McBurney, right rectus, and mid-line or paramedian. The latter incision was used when the diagnosis was in considerable doubt. For this particular analysis the cases surveyed were placed into two groups as indicated in Table III.

The McBurney incision was employed in 69 per cent and 87.9 per cent of the cases in the early and more recent periods, respectively. Simultaneously the use of the right rectus incision declined from 29.2 per cent to 11.0 per cent. The use of the mid-line incision remained about the same for both periods.

The comparison of the mortality rate with each of the three types of incisions during a given period was as follows. During the 1928–1932 and 1937–1938 periods the McBurney incision showed a mortality rate of 19.2 per cent in patients with appendiceal peritonitis, the right rectus incision was associated with a mortality rate of 23.3 per cent and the mid-line incision with a mortality rate of 45.5 per cent. During the more recent era (1944–1945) the McBurney incision revealed a mortality rate of 3.4 per cent, the right rectus

TABLE III

INFLUENCE OF THE CHOICE OF INCISION UPON THE MORTALITY RATE OF ACUTE APPENDICITIS. THE McBURNEY INCISION WAS ASSOCIATED WITH THE LOWEST MORTALITY RATE, THE RIGHT RECTUS INCISION WAS HIGHER, AND THE MID-LINE INCISION WAS HIGHEST OF ALL; "THE CLOSER TO THE MID-LINE THE INCISION IS MADE, THE HIGHER THE MORTALITY RATE." THIS DIFFERENCE WAS AS APPARENT IN THE PRE-CHEMOTHERAPEUTIC ERA AS IT IS NOW



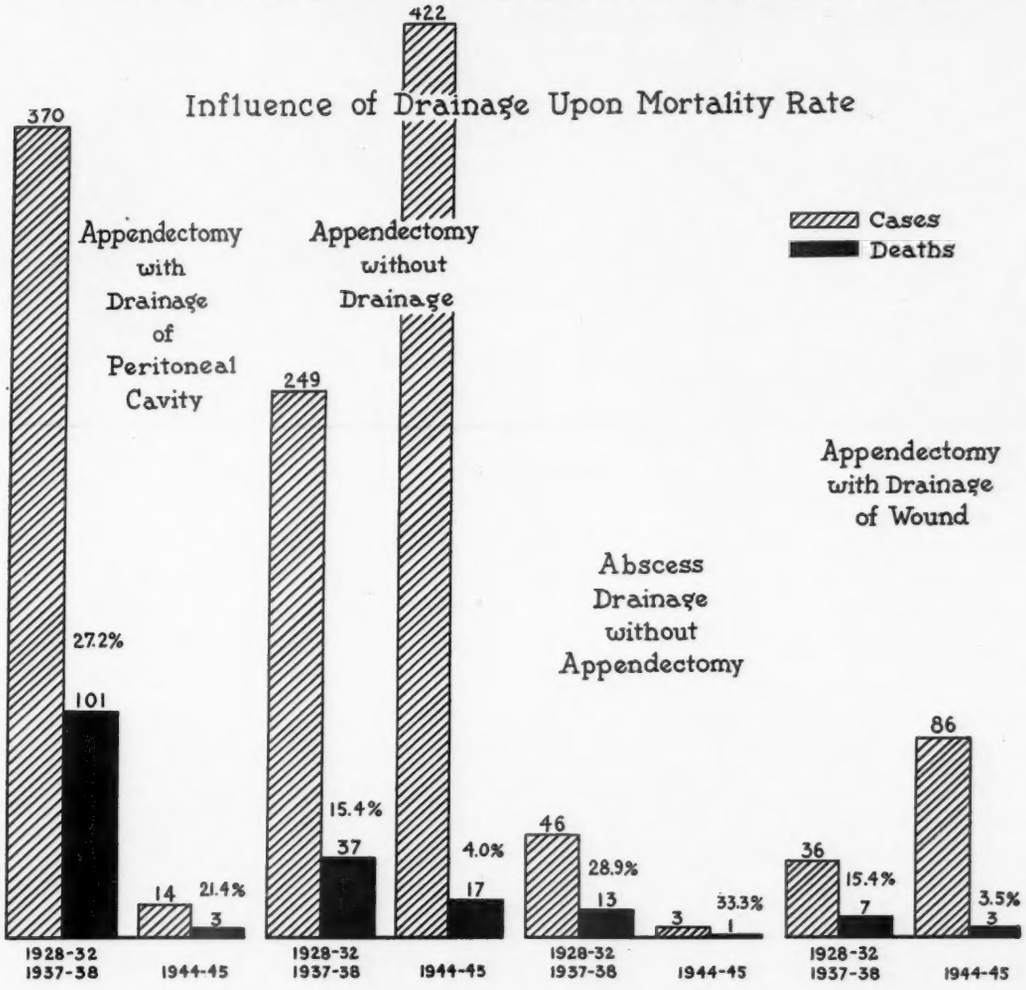
showed 12.0 per cent and the mid-line incision was 16.6 per cent. The final mortality rate differs in that it includes patients with far advanced appendiceal peritonitis never operated upon.

Influence of Drainage upon Mortality Rate. Table IV is presented with the purpose of illustrating the results obtained with appendectomy with and without drainage of the peritoneum, drainage of the peritoneum without appendectomy, and appendectomy with drainage of the wound down to the peritoneum. Again, the material has been divided between the years 1928–1932 (Period A) and 1937–1938 and the years 1944–1945 (Period B).

During Period A appendectomy with drainage of the peritoneal cavity for appendiceal peritonitis was employed in 370 cases with a mortality rate of 27.2 per cent. Appendectomy without peritoneal drainage was carried out in 249 such cases with a mortality rate of 15.4 per cent. An acute appendiceal mass was encountered

TABLE IV

INFLUENCE OF DRAINAGE UPON THE MORTALITY RATE OF ACUTE APPENDICITIS. PERITONEAL DRAINAGE WAS ASSOCIATED WITH THE HIGHEST MORTALITY RATE; DRAINAGE OF THE WOUND PRODUCED A MORTALITY RATE SLIGHTLY LESS THAN NO DRAINAGE AT ALL, BUT THE INCIDENCE OF WOUND INFECTION WAS LESS. CHEMOTHERAPY HAS NOT ALTERED THIS TREND.



in forty-six cases during this period and the operation was completed by insertion of a drain without appendectomy; this procedure carried with it a mortality rate of 28.9 per cent. During this same early period appendectomy for appendiceal peritonitis was performed and a drain was placed *down to* but not through the peritoneum and this resulted in a mortality rate of 15.4 per cent.

During Period B (1944–1945) appendectomy with peritoneal drainage was done in only fourteen patients and proved to have a mortality rate of 21.4 per cent. Appendectomy without peritoneal drainage was performed in 422 patients with a 4 per cent mortality rate. Drainage of an abscess without appendectomy was done in only three patients with a death in one. Appendectomy with insertion of a drain down to the sutured peritoneum was executed in eighty-six patients with an attendant mortality rate of 3.5 per cent.

Influence of Chemotherapy upon Complications of Acute Appendicitis. During the years 1944 and 1945 it was routine practice with our surgical house staff to employ sulfonamides intraperitoneally in the wound, and parenterally when a postoperative peritonitis was apparent. Sulfanilamide crystals were used locally, sodium sulfathiazole given intravenously was the choice for parenteral use. Penicillin was also used in a limited number of patients but these

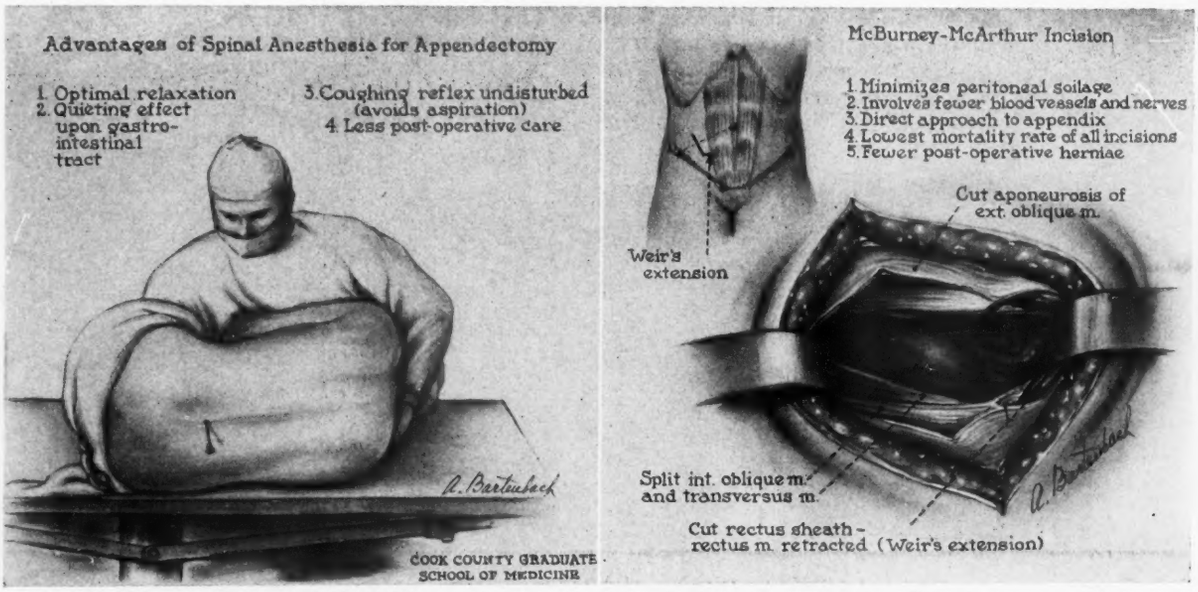

Advantages of Spinal Anesthesia for Appendectomy
1. Optimal relaxation
2. Quieting effect upon gastro-intestinal tract
3. Coughing reflex undisturbed (avoids aspiration)
4. Less post-operative care
COOK COUNTY GRADUATE SCHOOL OF MEDICINE
McBurney-McArthur Incision
1. Minimizes peritoneal soilage
2. Involves fewer blood vessels and nerves
3. Direct approach to appendix
4. Lowest mortality rate of all incisions
5. Fewer post-operative herniae
Cut aponeurosis of ext. oblique m.
Weir's extension
Split int. oblique m. and transversus m.
Cut rectus sheath - rectus m. retracted (Weir's extension)
1
2

FIG. 1. The anesthetic of choice in the treatment of acute appendicitis.
FIG. 2. The incision of choice in the treatment of acute appendicitis.

were the more severe cases of generalized peritonitis and probably cannot be used for purposes of analysis.

Purulent wound infections were seen in 120 of the 592 patients treated with sulfa drugs; specifically, sulfanilamide was placed into the wound of sixty-nine of these patients. In eighty of the patients with wound infection a drain was not placed either into the wound or the abdominal cavity whereas in the remaining forty wound infections a drain was used, most frequently down to the sutured peritoneum.

In spite of intensive chemotherapy, abscesses were still encountered as a complication. These were pelvic in eight patients, sub-hepatic in four and intra-abdominal in two.

Wound evisceration occurred in three patients operated upon during 1944 and 1945. One of these occurred through a McBurney incision, one through a right rectus, and the third through a right paramedian. The latter patient expired.

Remaining complications consisted of a massive atalectasis in two patients and a so-called postoperative pneumonia in eight.

OPERATIVE PRINCIPLES

Choice of Anesthetic. The ideal anesthetic agent for an appendectomy performed in the presence of acute inflammation would be one which disturbs the patient's immunologic processes the least, produces maximal relaxation, has a quieting effect upon the motility of the gastrointestinal tract and introduces the fewest postoperative complications. We believe that spinal anesthesia fulfills these criteria the best. We believe that a conscious patient is less apt to aspirate gastric content. Convulsions not infrequently seen under inhalation anesthesia of septic patients is less apt to occur under spinal anesthesia. In the very young and the very old it may be necessary to substitute general anesthesia, but even here induction with intravenous sodium pentothal is a worth while adjuvant. We have found the intrathecal introduction of novocaine or metycaine in doses averaging 1 mg. per pound of body weight (up to 150 mg.) produces approximately sixty and ninety minutes of anesthesia, respectively. This is given in the lateral recumbent position (Fig. 1) usually at the level of the third lumbar interspace. The total amount of drug is usually diluted up to 3 cc. with cerebrospinal fluid. To procure a satisfactory level of anesthesia the patient is placed in slight Trendelenburg position until the desired level is reached. Inasmuch as pain sensation re-

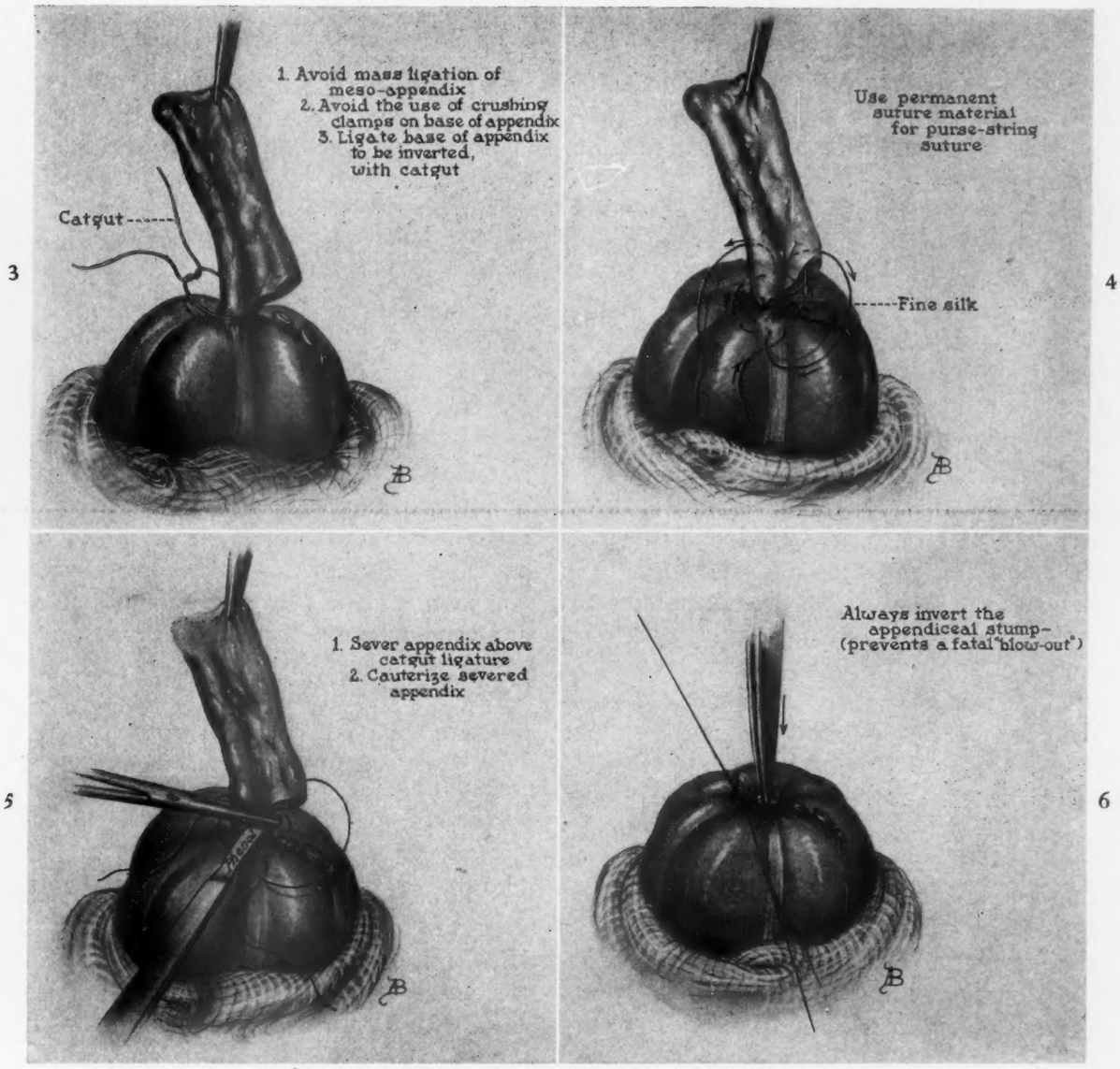

1. Avoid mass ligation of meso-appendix
2. Avoid the use of crushing clamps on base of appendix
3. Ligate base of appendix to be inverted, with catgut
Catgut
3
Use permanent suture material for purse-string suture
Fine silk
4
1. Sever appendix above catgut ligature
2. Cauterize severed appendix
5
Always invert the appendiceal stump—(prevents a fatal "blow-out")
6

Fig. 3. The management of the meso-appendix and base of the appendix.
Fig. 4. Method of purse-string suture less important than the use of a permanent suture material for that purpose.
Fig. 5. Chemical cauterization of the severed appendiceal stump.
Fig. 6. Inversion of the base of the appendix—a "must."

turns long before motor sensation, the operation can frequently be continued under intravenous sodium pentothal should the "spinal" anesthetic time be exceeded.

Choice of Incision. The McBurney-McArthur muscle splitting incision has quite arbitrarily been favored. (Fig. 2.) This incision was employed in 87.9 per cent of the cases since 1937 by our resident staff and in private practice it has probably been even more popular. We consider the following to be the advantages of this incision: (1) The appendectomy can be carried out with a minimal of peritoneal soilage. (2) There is less destruction of nerves and blood vessels than in the right rectus or battle-door incision. (3) This incision is more apt to place one directly over the appendix if it is made with the point of maximal tenderness in mind. (4) If the appendix is retrocecal, lateral extension of the incision will expose the lateral posterior wall of the cecum whereas upward extension of the battle incision would tend to be medial to the site of the pathological condition. (5) If the disorder lies

within the pelvis, Wier's extension (Fig. 2) into and along the lateral edge of the rectus provides adequate exposure. (6) It is associated with a lower incidence of postoperative hernia because of its anatomical nature.

Management of Meso-appendix. Mass ligation of the mesentery of the appendix was discouraged. McNealy has reported how such mass ligation distorts the ileocecal juncture and predisposes to subsequent obstruction of the terminal ileum. In acute appendicitis the mesentery is frequently indurated, friable and edematous, and reliance upon a single ligature is fraught with danger of subsequent hemorrhage. We believe further that sutures such as silk or cotton are apt to cut through this type of mesentery and, therefore, prefer fine chromic catgut as a series of interrupted ligatures for this purpose. (Fig. 3.)

Management of the Appendiceal Stump. Although every method of dealing with the base of the appendix is open to some theoretical objection, we have found the following principles to bear the test of experience. We believe in ligating the base of the appendix *without* application of a crushing instrument (Fig. 3); this avoids the danger of spillage and at the same time the danger of a slough due to pressure necrosis. The divided appendiceal stump is cauterized with phenol (iodine or electrocautery are equally acceptable). We have learned to consider inversion of the stump an obligatory part of the procedure. (Fig. 6.) It is the only way we know of preventing fatal fecal peritonitis due to a "blow-out." This we have witnessed time and again at necropsy following appendectomies for "interval" as well as acute appendectomies. The inversion should be carried out with a purse-string suture of some permanent material as advocated by Richter. (Fig. 4.) The exact modification of the purse-string suture that the individual operator wishes to use, we believe is unimportant, but that the material be a permanent one we are of the opinion is important. Whether the base of the cecum is involved should not be a criterion, for then the operator should be placing his sutures far enough out into the cecum so that they are anchored to viable tissues. Finally, some form of peritonealization should be carried out (Fig. 7) whether it be by simply bringing over an ileocecal fat pad or a series of seromuscular sutures of the cecum. This will do much to prevent mechanical bowel obstruction from adhesions.

Drainage. The insertion of drains into the peritoneum is based upon the fallacious belief that such foreign bodies will siphon away exudate. Actually, within a matter of hours drains are so well walled off by fibrinous exudate that their function is soon lost, and instead of a drain we are left with a plug. We have long believed and have taught that the outcome of the operation was dependent more upon the degree of peritonitis than upon the type and number of drains inserted into the abdomen. Accordingly we have discouraged its use.

Drainage of the wound is a different matter. (Fig. 8.) Here extensive soilage is very prone to occur and the defense mechanisms are overbalanced by rather favorable culture media provided by extravasated blood, serum, fat and catgut sutures. Wound infections are, therefore, a frequent complication and it still appears that the most effect prophylaxis to that is a small drain inserted down to the sutured peritoneum. (Fig. 8.) Such a drain can be removed at the time the sutures are removed and does not delay convalescence.

Local Chemotherapy. The majority of the patients were treated in 1944 and 1945 by the application of varying amounts of sulfanilamide into the ileocecal area, the wound, and into the general peritoneal cavity when operative conditions permitted. Inasmuch as this was almost always supplemented by subsequent parenteral chemotherapy, it is difficult to appraise the value of its local use. However, it is difficult to consider 5 or 10 Gm. of sulfanilamide, locally applied, as self-

sufficient. Furthermore, in view of the inactivation of sulfanilamide by purulent exudate, the actual value of intraperitoneal and wound application is questionable. For this reason it might be well to use penicillin intraperitoneally because exudate does not affect it and a single dose will provide blood levels for five to six hours which are higher than equivalent doses given intravenously or intramuscularly.[2] However, the intraperitoneal use of penicillin awaits further experimental work.

Suture of the Wound. Our method of closure of an appendectomy wound is identical to that used in other abdominal wounds. The peritoneum is closed with fine interrupted silk on atraumatic needles. The internal oblique is loosely approximated with interrupted No. 32 stainless steel wire (Fig. 9) and the aponeurosis of the external oblique with the same type of suture, again interrupted. The skin is sutured with interrupted sutures of silk, dermal or nylon.

The use of permanent sutures is considered important for several reasons. There is much less exudation of serum and polymorphonuclear leucocytes than occurs with catgut; this we know minimizes chances for purulent exudation. Most infections of the wound are superficial to the aponeurosis of the external oblique or rectus fascia and wire sutures hold the tissues tight in the presence of purulent infection; we have rarely seen wire sutures extruded from a wound. In fact, on opening an occasional wound abscess we have been able to visualize the wire suture holding the fascia firmly together in the midst of the inflammatory exudate. Wire does not draw exudate through a tract as silk does for it is a solitary cylinder and not a wick composed of several twisted strands thereby acquiring the power of capillary attraction.

The use of permanent interrupted sutures further facilitates early ambulation which we employ wherever possible. By this we mean getting the patient out of bed at least twice on the first postoperative day and for increasing time intervals thereafter.

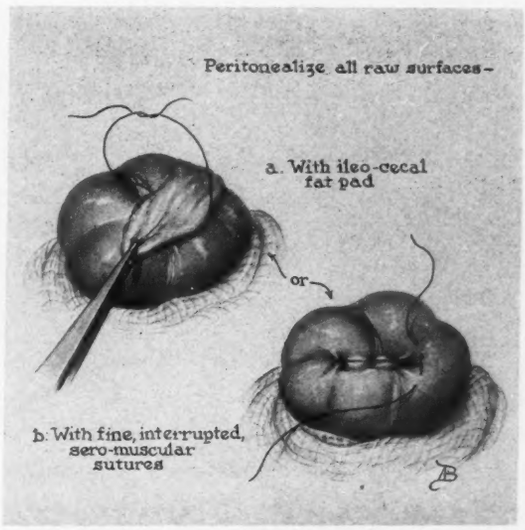
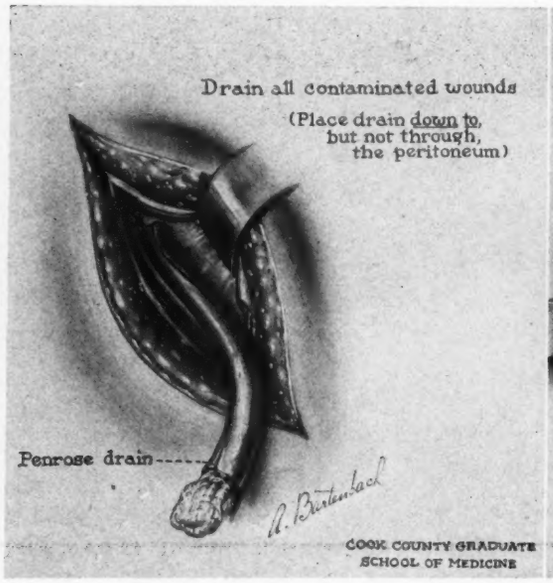


FIG. 7. Peritonealization of all raw surfaces.

The advantages of this are many and constitute an independent report[3] but suffice it to say here that patients who do not have a generalized peritonitis, appendiceal abscess or ileus are permitted ambulation the next day after appendectomy. We consider this method of wound closure essential for that purpose.

Postoperative Requirements. (1) Patients suffering from appendiceal peritonitis receive a minimum of 3,000 cc. of parenteral fluids per day plus whatever additional is lost through intragastric suction. Part of this fluid intake should supply protein either in the form of whole blood, plasma or amino acids. Not more than 1,000 cc. of this amount of fluid should be saline unless there is marked losses due to suction. The fluid intake should be sufficient to insure a twenty-four-hour urine volume for the adult of not less than 1,000 cc. with a specific gravity less than 1.015.

(2) Intragastric suction (we favor the Levine tube) is an important adjuvant in the treatment of appendiceal peritonitis. Its chief purpose is to prevent or decrease distention, its effect is explained by the important observation that the bulk of intestinal gases comes from swallowed atmospheric air. Such suction is maintained until signs of returned peristalsis is apparent as judged by active bowel sounds, passing of flatus, bowel move-


Drain all contaminated wounds
(Place drain down to, but not through, the peritoneum)
Penrose drain
COOK COUNTY GRADUATE SCHOOL OF MEDICINE

8

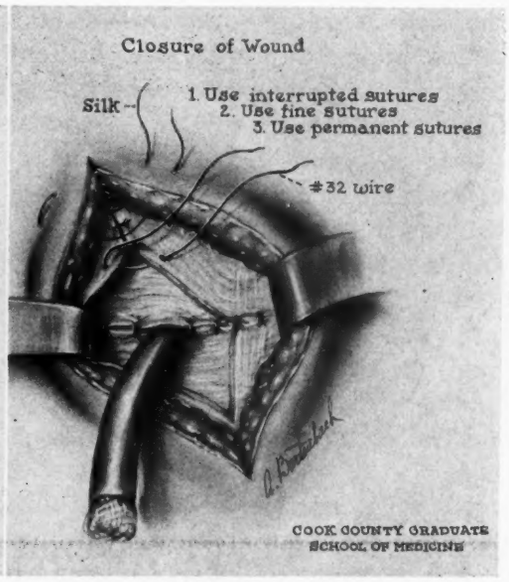


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FIG. 8. Insertion of a cigarette drain down *to* the peritoneum decreases wound infections.
FIG. 9. Use of fine permanent sutures throughout all layers in closure of the wound.

ments or actual demonstration of a "positive pyloric balance" when a careful record of fluids taken in per os and recovered by suction are compared. This usually occurs on the third or fourth postoperative day. We have rarely resorted to the use of the Miller-Abbott tube to treat the ileus of appendiceal peritonitis; however, its use in the decompression of a mechanical obstruction complicating an appendectomy may be crucial and avoid a second operation.

(3) Chemotherapy is a routine measure in the treatment of appendiceal peritonitis. We have utilized either sodium sulfathiazole or sodium sulfadiazine intravenously. To maintain a blood level between 8 and 12 mg. per cent from 2½ to 5 Gm. per day are sufficient. With the use of sulfadiazine in preference to sulfathiazole, and careful attention to renal output, we have rarely encountered a sulfa anuria. More recently penicillin has been made available and it has been used in conjunction with sulfonamide therapy rather than in place of it. Its effect is yet to be evaluated.[4]

(4) Oxygen inhalation, using the B.L.B. mask to achieve 100 per cent concentrations has been used for the more severe cases of peritonitis. The indications and benefits are manifold: the anoxemia, tachycardia, shock and bowel distention are all features of generalized peritonitis which suggest oxygen therapy.

(5) Intravenous vitamin therapy is indicated by virtue of the depletion of vitamins B and C by such an acute febrile illness, to improve the metabolism of carbohydrate administered by vein, and to promote more satisfactory wound healing.

(6) We *do not* administer peristaltic stimulants such as prostigmine, pitressin, pilocarpine or irritating enemas. An ileus is a physiological response to a peritonitis which will respond spontaneously as soon as the peritonitis is brought under control. The use of drugs or enemas is very much like whipping a tired horse, if one may use such a homely anology.

(7) The treatment of the intra-abdominal complications of acute appendicitis cannot be retold here, but has been described previously by one of us.[5] These require far greater skill and judgment than the removal of the appendix and it is well for the attending surgeon to be familiar with these complications for we doubt that the era of chemotherapy will entirely prevent them.

COMMENTS

Although we have been teaching the principles of the treatment of acute ap-

pendicitis to our house staff long before such data as we have accumulated here were available, it remains to be seen whether our statistics justify the position we have taken. Let us list these principles again and see what experience within one institution has shown.

Most important in the treatment of appendicitis is the performance of the appendectomy prior to perforation. It is apparent from our own data (Table I) that the improvement in the mortality rate for the treatment of acute non-perforated appendicitis is not statistically significant. In other words, if we could be certain that all acute appendicitis would reach us prior to perforation, we could assure the patient a mortality rate well under 1 per cent without recourse to any of the modern therapeutic adjuncts available. This obviously is as strong an argument for early appendectomy as we need. Nevertheless, we noted that in 1945, approximately 40 per cent of these patients had taken cathartics prior to admission; and better than one-fourth of the appendices had perforated!

The most striking improvement in the treatment of appendicitis has occurred in the treatment of appendiceal peritonitis. For in a period of approximately fifteen years the mortality rate from acute appendicitis with perforation has dropped from 26.4 per cent to 13.9 per cent, a decrease of almost one-half. Inasmuch as the operative technic for the removal of a perforated appendix is obviously the same as for a non-perforated appendix, the explanation for the marked improvement in the mortality rate of the former lies in the postoperative care and not surgical technic. Furthermore, inasmuch as the greatest improvement in the treatment of appendiceal peritonitis occurred since 1938, when blood was already available to our patients, it would appear that the greatest factor was chemotherapy. The benefits of chemotherapy in the treatment of appendicitis has adequate confirmation.[6–13]

Another teaching principle has been to avoid operative intervention in the patient with an acute appendiceal abscess based upon the concept that such a patient is localizing his inflammation well and that the leak from the intestinal tract will be sealed by this means. Operation in this type of case may result in what has been well termed an "induced" peritonitis. Analyzing the record (Table II) one observes that the former mortality rate of 18 per cent for operated cases was reduced to 4.2 per cent when conservative measures were utilized; more recently, in spite of chemotherapy, the mortality rate in such cases operated upon was 8.5 per cent whereas those without surgery was 3.1 per cent. Presumably non-surgical intervention rather than chemotherapy is the answer to the management of the patient with an acute appendiceal mass. The value of this precept has progressed to the point where cancelling the operation is advised if a mass becomes more definitely palpable under anesthesia, or simply draining the abscess if one is inadvertently encountered on opening the peritoneum.

The McBurney-McArthur muscle splitting incision has been favored for a number of anatomical reasons. Both in the periods prior to 1938 and more recently, this incision has been associated with a significantly lower mortality rate than any other. In fact we have used the maxim that the closer to the mid-line the appendectomy is performed, the higher the mortality rate. While it is true that right rectus and midline incisions are utilized more often when the diagnosis is in doubt, yet this alone cannot account for a recent mortality rate with the McBurney-McArthur incision approximately one-fourth of either of the other two. If one remembers that one can extend this incision medially to achieve the advantages of the right rectus incision, then certainly there remains little advantage in exposing a patient to the increased mortality risk.

Although we have presented other evidence discouraging the use of intraperitoneal drains, undoubtedly its influence upon

the mortality rate will be the most convincing. Here we found, in every period studied, the highest mortality rate for patients with peritoneal drains, considerably lower mortality rates for those not drained, and still lower mortality rates for those with a drain placed down to the sutured peritoneum. We acknowledge the greater tendency to place peritoneal drains in patients with the more severe grades of peritonitis, but in 1944 and 1945 the majority of the patients with appendiceal peritonitis did not have intraperitoneal drains and their was a marked lowering of the mortality rate whereas the few who did have such drains inserted showed little improvement in the mortality rate. The value of inserting drains into the wound (not the abdominal cavity) is further attested by the fact that two-thirds of the patients with wound infections did not have wound drainage.

SUMMARY*

1. The mortality rate for the removal of acute non-perforated appendices was well under 1 per cent, without significant improvement in recent years.

2. A lowering of the mortality rate from 26.4 per cent to 13.9 per cent has occurred in the treatment of acute perforated appendices with appendiceal peritonitis; this is attributed chiefly to chemotherapy.

3. The greatest improvement in the treatment of acute appendiceal abscesses occurred from non-operative intervention.

4. The McBurney-McArthur muscle-splitting incision is associated with the lowest mortality rate; the closer the incision is made to the mid-line, the higher the mortality rate.

5. Appendectomy with drainage of the peritoneal cavity was associated with the highest mortality rate.

6. Appendectomy without drainage of the peritoneal cavity showed a much lower mortality rate than similar cases which were drained.

7. Appendectomy with drainage of the wound (usually for a perforated appendix) showed a mortality rate slightly lower than appendectomy without drainage but a much lower incidence of wound infections.

8. The surgical principles thought to be of value in the treatment of acute appendicitis are discussed in full detail.

* In a recently completed series of 108 patients operated upon for acute suppurative appendicitis and treated prophylactically with penicillin at the Cook County Hospital, Drs. Griffin, Silverstein, Hardt and Seed[16] report only one death. This represented a marked improvement over a 4 per cent mortality rate reported by them for all patients operated upon. Our statistics included all cases, both operated and non-operated. The latter type is frequently moribund on admission and materially affects the mortality rate.

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ACUTE SURGICAL CONDITIONS OF THE ABDOMEN: CONGENITAL LESIONS

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IN any consideration of congenital lesions it should be remembered that anomalies tend to be multiple, and when one is found thorough search should be made for others which may be more serious. In 15 per cent of all types of congenital malformations multiple major defects exist which are not amenable to surgical treatment and the prognosis is hopeless.[2] The majority of serious maladies of early infancy are due to mechanical lesions or congenital lesions of a mechanical nature rather than any histopathologic alteration such as neoplasia or a primary inflammatory process.

The following discussion omits many congenital lesions of the abdomen as they do not constitute an acute surgical condition.

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

Congenital hypertrophic pyloric stenosis is the most common acute surgical condition of the newborn. The cause of this interesting disease still remains unknown; however, there seems to be an hereditary factor as it occasionally occurs among two or more members of the same family. The ratio of males to females affected is 4:1. There is hypertrophy of the circular muscle of the pylorus with an actual increase in the number of muscle fibers. This pyloric musculature is of variable size, but it usually forms a smooth, bulbous mass about 2 by 1.5 cm. The stenosis is caused by the large, firm, unyielding mass of muscle compressing the mucosa and greatly decreasing the lumen in this region.

Symptoms. Vomiting usually begins when the infant is about two weeks old, seldom before. It begins gradually but soon becomes forceful and projectile and occurs after most feedings. As the obstruction is above the papilla of Vater, the vomitus does not contain bile. This is important in differentiating other obstructive lesions. The child usually is not seen until four to six weeks of age. By then the amount of stool has decreased, and with continued decrease of fluid and caloric intake, there are loss of weight and dehydration. Wrinkling of the skin and loss of elasticity may be marked. Electrolytic imbalance may quickly supervene with alkalosis leading to tetany.

Physical findings. A small mass almost always can be palpated in the right upper quadrant when the abdomen is relaxed. Relaxation can be produced by laying the baby on his abdomen and gently palpating with the hand under the infant. Feeding the baby often will relax the abdomen and there is usually a brief period of relaxation immediately after vomiting.

Roentgenologic examination reveals a large, gas filled stomach with little gas in the intestine. It is most rare that barium studies are indicated.

Differential diagnosis. Congenital hypertrophic pyloric stenosis must be differentiated from pylorospasm in which the vomiting tends to occur in spells and is not progressive. There is often associated hypertonicity with pylorospasm and the symptoms can be relieved by atropine. Unsuitable formulas that are too large or too concentrated may cause postprandial regurgitation, which may be confused with pyloric stenosis.

Stenosis or atresia of the duodenum or intestine usually produces emesis very soon after birth. The vomitus in such cases

usually contains bile. Atresia or stenosis of the esophagus may be associated with tracheoesophageal fistula with resulting respiratory complications.

Incomplete rotation of the intestine, which obstructs usually at the duodenum below the papilla of Vater, produces vomiting within the first twenty-four to forty-eight hours after birth. The vomitus contains bile.

Intracranial hemorrhage or traumatic injury occurring at birth or later may cause emesis but this is not necessarily associated with feeding and occurs at irregular intervals. Other neurologic signs usually are present to aid the differentiation.

Treatment. Although congenital hypertrophic pyloric stenosis is an acute surgical condition, operation should be delayed until the child's nutritional and electrolytic state is improved as much as possible. This often takes two or three days but the time is well spent. Glucose should be given intravenously in 5 per cent or 10 per cent concentration, 10 cc. per pound of body weight being given twice daily, and physiologic saline solution should be given by hypodermoclysis in amounts up to 15 cc. per pound of weight. Thickened feedings should be given during this preparatory period. Atropine may be used, but is of questionable value.

Technic. Anesthesia should be sufficient so that the abdomen is relaxed during the entire operation. Drop ether is the anesthetic agent of choice and affords relaxation with a good margin of safety.

Through a right rectus incision the pyloric region is exposed and the bulbous mass is held between the thumb and index finger. A longitudinal incision is made through the muscle on the anterior surface down to the mucosa. The muscle then is separated more with a small hemostat until the mucosa bulges between the cut edges of the muscle. When the muscle is incised it should be done with great care as the muscle at the distal or duodenal end of the bulbous mass very abruptly thins out and consequently the duodenal mucosa can be entered very easily. If the mucosa is incised it should be closed and covered with a piece of omentum.

Closure of the abdomen should be done accurately and carefully. The tissues often are like spider web and not much stronger. Disruption can occur even two or more weeks after closure. It is best to use fine interrupted sutures and several through-and-through retention sutures passing through the skin, muscle and peritoneum.

Postoperative care. The child may be fed soon after consciousness is regained. One ounce (30 cc.) of fluid should be given every two hours starting with water for four or five feedings and half-strength formula for four or five feedings; the strength and amount of formula then should be increased gradually so that the full formula is fed about forty-eight or sixty hours after operation.

In addition to these feedings it is best to give fluids parenterally for the first two or three days so that the child receives an over-all total of 3 ounces (90 cc.) of fluid for each pound of weight.

The relationship between fluid exchange and extracellular fluid in an infant weighing 7 kg. compared to an adult weighing 70 kg. is pictured in figure 1, taken from Gamble.[4] The relatively larger surface area of an infant prescribes a basal rate of heat production per kilogram which is approximately twice as rapid as in the adult. Therefore, insensible water loss per kilogram is twice as large. Minimal water expenditure per day by the infant is 300 cc. and by the adult is approximately 1,400 cc. Extreme dehydration thus can quickly develop in an infant with insufficient intake, especially in the presence of vomiting, when loss of electrolytes further complicates the fluid imbalance.

MECKEL'S DIVERTICULUM

Meckel's diverticulum is the vestige of the vitelline duct, which had opened into the ileum. A small cord of tissue occasionally connects the diverticulum to the umbilicus and very rarely a fistula is

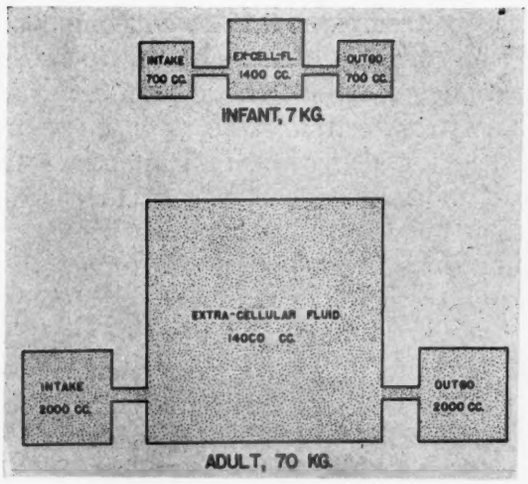

FIG. 1. The relationship of water exchange to extracellular fluid in an infant weighing 7 kg. and an adult weighing 70 kg. The rapidity with which dehydration can develop in an infant can be seen in this diagram. Intake of fluid in the infant is relatively large and amounts to half the total extracellular fluid. Intake in the infant also is one-third the intake of an adult weighing 70 kg. (From GAMBLE, J. L. Chemical anatomy, physiology and pathology of extracellular fluid: a lecture syllabus. Harvard Medical School. 4th ed. Cambridge, Massachusetts, 1944. Harvard University Press.)

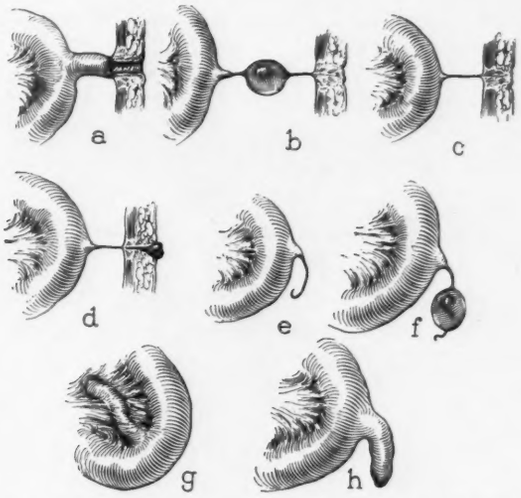

FIG. 2. Forms in which the remains of the vitelline duct may be found: *a*, patent duct opening onto the abdominal wall at the umbilicus; *b*, the duct occluded at its ends so that a cyst is formed somewhere in its length; *c*, the duct obliterated but remaining attached to the umbilicus; *d*, the duct remaining attached to the umbilicus and having intestinal mucosa at its outer end; *e*, the duct obliterated and hanging with a free end within the abdominal cavity; *f*, the duct hanging within the abdominal cavity and forming a cyst; *g*, the duct lying within the mesentery of the small intestine, and *h*, the duct remaining as a small, patent projection from the ileum. (From DIXON, C. F. and STEWARD, J. A. Diverticulum with hemorrhage; omphalo-adenoma. *S. Clin. North America*, 12: 1025–1031, 1932.)

present. Ladd and Gross[11] reported six cases of intestinal obstruction caused by a band running to the umbilicus. Figure 2 shows some of the forms in which the remains of the vitelline duct may be found.

Meckel's diverticulum occurs about twice as frequently in males as in females and usually arises on the antimesenteric side of the terminal ileum 1½ feet to 3 feet (46 to 91 cm.) proximal to the ileocecal valve. It varies in size from ½ inch to 5 inches (1.3 to 13 cm.). The lining epithelium contains gastric mucosa in about 50 per cent of the cases. Occasionally, duodenal, pancreatic or colonic tissue can be identified. This heterotopia accounts for some of the symptoms. Unexplained hemorrhage from the intestinal tract fairly frequently is from a bleeding peptic ulcer occuring in a Meckel diverticulum.

Symptoms resembling those of appendicitis can be caused by a diverticulum which may become inflamed and perforate. A Meckel diverticulum always should be looked for when a normal appendix is found with symptoms of acute appendicitis. Volvulus may occur around a Meckel diverticulum or the diverticulum may become twisted and infarcted. It also may be the leading point of an intussusception.

Figure 3 shows a Meckel diverticulum in an eleven year old boy which looped around the root of the mesentery forming a half-hitch with itself and obstructing the lower third of the ileum. The knot was so tight it was difficult to untie at the time of necropsy.

Treatment. If a normal appearing Meckel diverticulum is found which is small, is unattached to the abdominal wall or to other organs, and has an adequate lumen, it should not be removed. If it has produced symptoms and should be removed, it should be excised in such a manner as to leave an adequate lumen. This is usually accomplished by excision

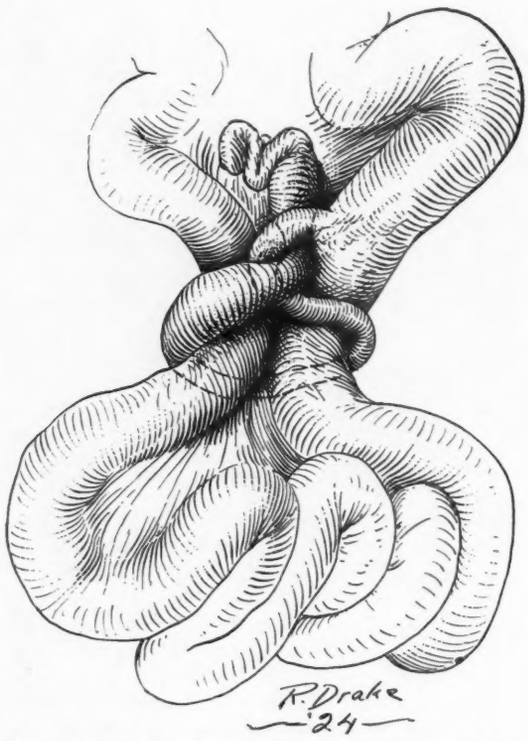

Fig. 3. A Meckel diverticulum looped around the root of the mesentery and forming a half-hitch with itself.

with an elliptical incision in the long axis of the ileum and suturing the transverse axis. Closure with a purse-string suture should not be attempted.

If an intussusception is present an attempt should be made to reduce it. If the symptoms are of more than thirty-six hours' duration, the intussusception should be fixed with interrupted sutures so that it cannot advance and a short-circuiting enterostomy or ileocolostomy should be carried out.[13] The invaginated portion then may slough out through the lumen of the intestine. The mortality rate is much lower if this procedure is used than if resection and anastomosis are performed in acute cases.

DUPLICATIONS OF THE GASTRO-INTESTINAL TRACT

Duplications of the gastro-intestinal tract are very rare but should be remembered and recognized as they may lead to acute surgical complications. Inclusion cyst, enteric cyst and enterogenous cyst are, in reality, duplications of some portion of the gastrointestinal tract, although they may not duplicate that part to which they are connected or attached. Duplications of the gastrointestinal tract are most frequently found in the small intestine. The structure has one or two muscular layers and is lined with mucosa. At times the lumen communicates with the lumen of the gastrointestinal tract. Lewis and Thyng[12] frequently found duplications of the fetal mammalian alimentary tract, which normally regress. These may represent pinched-off cells of the fetal intestinal tract. Triplication of the colon has been reported by Gray.[6]

The duplication usually contains clear fluid and is firmly adherent to the normal stomach, intestine or colon with no line of cleavage between the duplication and the normal organ. In removing such a structure, it is usually necessary to resect the adjacent normal portion of the gastrointestinal tract, as the duplication cannot be peeled or dissected out as can a mesenteric cyst, with which it may be confused.

Symptoms. The symptoms are usually those of obstruction, which may be mild and repeated over a long period or may be acute, occurring soon after birth. The symptoms are caused by encroachment on the normal intestinal lumen by accumulation of secretions in the blind duplication. This enlargement may occur between the leaves of the mesentery and impair the blood supply to the normal intestine.

Treatment. Occasionally an opening made between the duplication and the adjacent portion of the intestinal tract will adequately drain the part without later filling with food or intestinal contents. However, because of the size of the lesion, common blood supply, and lack of cleavage planes, resection of the duplication and adjacent gastrointestinal tract usually is necessary.

Griess, Dixon, Weber and Bargen[7] recently reported a case of complete duplication of the colon. The patient was a twenty-seven year old woman, who had been operated on elsewhere for intestinal obstruction at four years

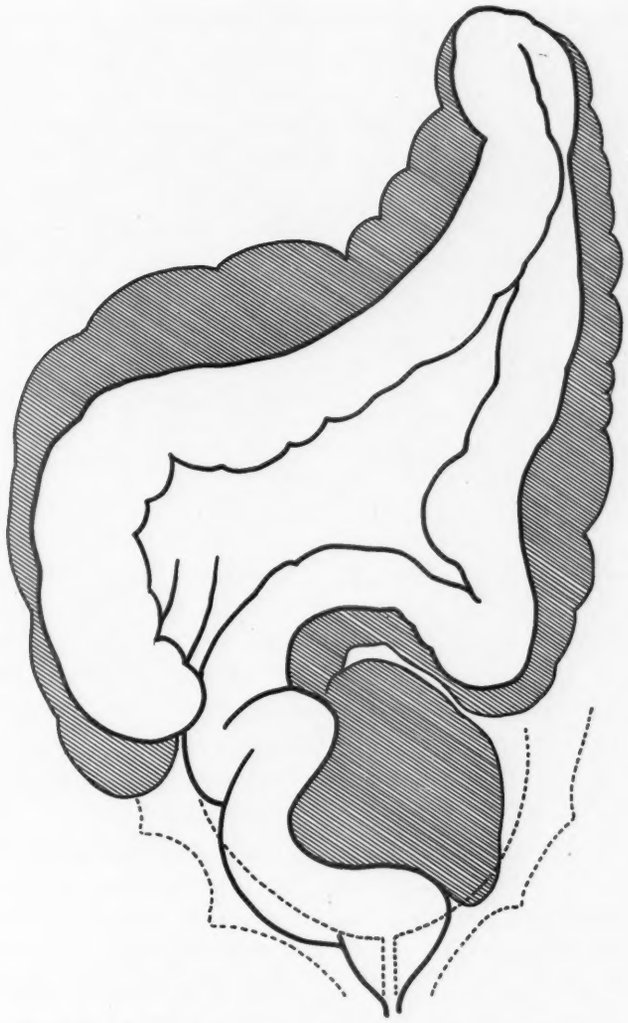

Fig. 4. Diagram of roentgenogram of colon showing radiopaque concretion in the blind duplicated colon. (From Weber, H. M. and Dixon, C. F. Duplication of the entire large intestine (colon duplex): report of case. *Am. J. Roentgenol.*, 56: 319–324, 1946.)

of age and twenty-three years of age. At the time of the last obstruction the surgeon drained what appeared to be a cyst. Two appendixes, one of which was gangrenous, were removed at twenty years of age.

The patient had had symptoms of partial obstruction for one month before the present admission. Physical examination revealed a double infantile uterus, a vaginal septum, and a pelvic mass. There were two draining fistulas in the scar in the left lower quadrant. Roentgenologic examination showed a complete duplication of all segments of the colon, including the cecum and rectum. (Figs. 4 and 5.) Both colons seemed to be complete morphologically, but the one without external communications was filled with what appeared to be a fluid, calcareous material. The rectum with the external communication was displaced by the mass composed of the other rectum.

At operation the patient was found to have congenital absence of the right kidney, two uteri and the double colon. The two colons arose from the same mesentery. The fistulas were removed and a communication was made between the two colons by resecting 8 or 10 inches (20 or 25 cm.) of each in the sigmoid region as an exteriorization operation. Later clamps were applied to the spurs and the colonic stoma was closed.

IMPERFORATE ANUS

As this symposium is limited to acute abdominal surgery, a complete review of imperforate anus will not be given.

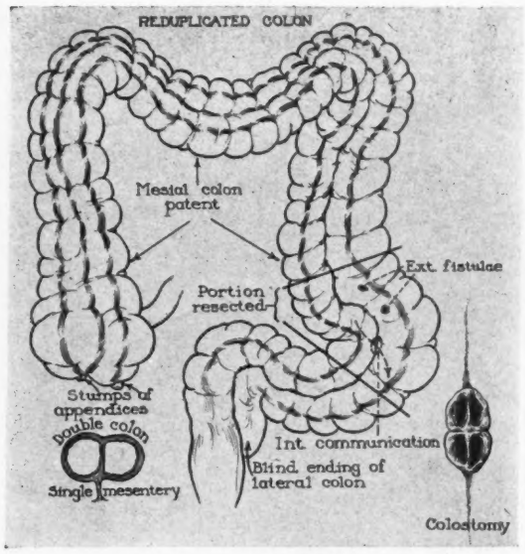

FIG. 5. Double colon, as found at operation.

It is important to determine the position of the blind rectal pouch, so that the proper surgical procedure may be decided on. Holding the infant head down as described by Wangensteen and Rice[17] with roentgenograms taken in both the lateral and the anterposterior position is very helpful. Roentgenograms taken within the first twelve to twenty-four hours after birth may not be satisfactory as the colon may not yet contain sufficient gas.

If there is an anal dimple which bulges when the infant cries, perforation of the separating membrane probably will establish continuity. If repair by the perineal route cannot be carried out, immediate abdominal reconstruction should not be attempted. Transverse colostomy can be done if there is a possibility of later pull-through procedure. However, it is well to have the stoma as near the end as possible because of the collection and inspissation of mucus in the blind loop when transverse colostomy is done.

FAILURE OF INTESTINAL ROTATION (MALROTATION)

During the embryonic development of the human intestine wide change in position takes place before the adult arrangement is obtained. In the 7.5 mm. embryo the umbilical loop has been formed and is herniated into the umbilical cord as a result of intra-abdominal pressure and increased length of the intestine. By the time the embryo is 10 mm. in length the umbilical loop has turned about 90 degrees to the right, that is, counterclockwise. The increase in length and extension of the colon so that the cecum occupies the right lower abdominal quadrant may not be finished until the first year of extra-uterine life.

Congenital anomalies associated with failure of intestinal rotation and capable of producing serious digestive disorders of a mechanical nature are known to exist. These anomalies may be divided into two groups: (1) the group in which there is failure of fixation of the small intestine, making possible volvulus of the entire mass of the intestine in a clockwise or counterclockwise direction about the mesenteric pedicle and (2) the group in which there are persistent congenital mesenteric bands producing intermittent or absolute intestinal obstruction.

During the development of the embryo mesenteric bands have a definite normal function in the rotation of the intestinal tract. Wakefield and Mayo[16] have shown that the maintenance of these mesenteric bands after birth may produce intestinal obstruction. (Figs. 6, 7 and 8.) The complexity of rotation can be visualized by study of the intestine of the cat. Figure 9 shows the arrangement of the intestine and mesentery in the cat comparable to that of the human embryo before rotation begins within the abdominal cavity. The mesentery between the closely approximated segments of intestine (A and B) is the duodenocolic isthmus. The mesenteric artery is on the left side of the vein and the duodenum is intraperitoneal. In Figure 10 the midgut has started its rotation about the duodenocolic isthmus (A and B), which is now in the vertical axis. In figure 11 the final arrangement is seen, as is found in the normal adult human. The small intestine now occupies the left side of the abdomen and the portions indicated by A and B have changed from right to left and from

Fig. 6. Congenital mesenteric bands causing duodenal obstruction in a woman forty-two years old. There is malrotation of the colon and the duodenum is intraperitoneal. (From Wakefield, E. G. and Mayo, C. W. Intestinal obstruction produced by mesenteric bands, in association with failure of intestinal rotation. *Arch. Surg.*, 33: 47–67, 1936.)

left to right respectively. The ileum now enters the colon from the left and the descending portion of the duodenum is dorsal to the colon. The superior mesenteric artery, which formerly occupied the duodenocolic isthmus, now passes anterior to the duodenum and is on the right side of the vein.

Figure 6 shows the mesenteric bands found in a forty-two year old woman who had had repeated attacks of abdominal pain and vomiting since childhood. The duodenum was found to be intraperitoneal and angulated by traction bands. The transverse colon was found to be attached to the dorsal portion of the parietal peritoneum in the upper part of the abdomen.

Figure 12 shows the small intestine twisted around an abnormally placed colon. The unusual length and mobility of the mesentery of the small intestine combined with the abnormal position of the colon, resulted in volvulus. The inset shows the colon in the left side of the abdomen and the intraperitoneal position of the duodenum.

Gardner and Hart[5] reported 105 cases collected from the literature of anomalies of the intestinal tract causing obstruction. In eighty-eight of the 105 cases the condition was the result of volvulus of the entire mesentery. Ladd and Gross[11] have called attention to the importance of recognizing the obstruction of the descending duodenum that usually is present with volvulus of the midgut or entire mesentery. This duodenal obstruction is caused by an overlying cecum or a persistent mesenteric band. Unless this is severed, the duodenal

FIG. 7. Malrotation of the colon, which occupies the left side of the abdominal cavity. There is a persistent congenital mesenteric band obstructing the duodenum. The duodenum is entirely intraperitoneal. Search always should be made for this congenital band after one has reduced a volvulus of the small intestine associated with malrotation of the colon.

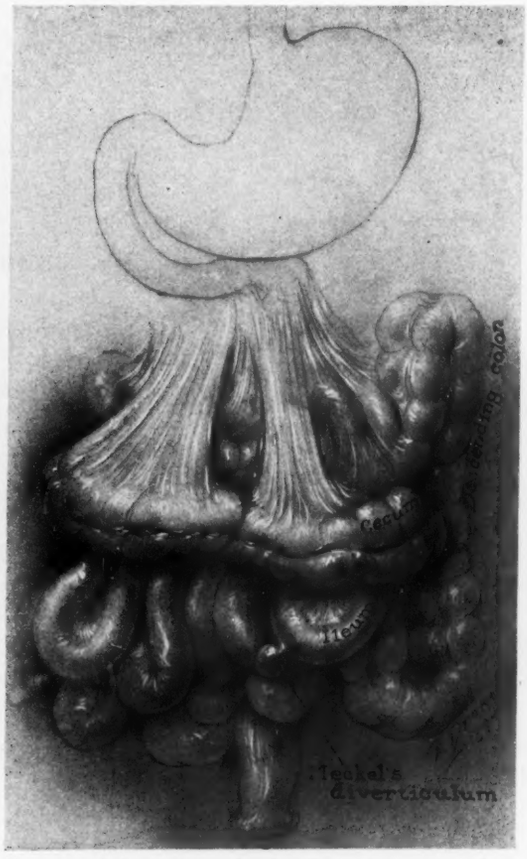

FIG. 8. Malrotation of colon in man twenty-four years old. The malrotation has produced intermittent obstruction of the small bowel since infancy.

obstruction persists even though the volvulus has been corrected. (Fig. 7.)

Symptoms. Many cases of incomplete rotation are asymptomatic and may be found only incidentally during roentgenologic examination or at the time of an abdominal operation.

Symptoms, if present, are those of obstruction and depend on the location and degree of obstruction. In the majority of cases the infant is seen within the first two or three months after birth because of vomiting due to duodenal obstruction. The obstruction is usually partial and below the papilla of Vater so that bile is present in the vomitus and there is passage of stool.

Dehydration occurs rapidly and may be severe. If volvulus occurs there may be considerable distention from entrapped gas. The temperature may reach 104° to 105°F. from either the dehydration or infarction associated with a volvulus.

Barium studies are usually unnecessary and undesirable in acute cases because of the danger of aspiration into the lungs or inspissation in the intestine. In older children and adults with recurrent attacks of obstruction or vague abdominal symptoms, studies by barium enema and barium by mouth should be carried out.

Ladd and Gross[11] cited a case of a twenty-one month old boy with symptoms of several months of recurrent attacks of abdominal swelling associated with vomiting or diarrhea diagnosed as celiac disease. In reality the symptoms were due to volvulus of the entire midgut. In their opinion all persons who have celiac syndrome should undergo barium studies to detect any malrotation, as surgical correction of the latter may relieve the patient's symptoms.

Treatment. Adequate preparation with fluids, electrolytes and blood if necessary should be carried out in all cases. A long right rectus incision to afford good exposure should be made. In an infant an obstruction of the duodenum due to a mesenteric

band often will be found. This is usually associated with a volvulus of the entire mesentery.

If the right half of the colon is not seen on opening the abdomen but only distended and probably bluish, discolored small intestine, a volvulus of the midgut should be recognized. It is easily mistaken for a herniation of small intestine through a portion of mesentery. The incision should then be enlarged, if necessary, and the small intestines delivered onto the abdomen where the true nature of the process can be seen. The volvulus is usually clockwise and should be unwound before the intestines are replaced. The mesenteric band compressing the duodenum, which almost always is present, should be severed, as closure at this time probably will result in death unless the band is cut. No attempt should be made to rotate the bowel to its normal position, but the right half of the colon should be allowed to rest in the left side of the abdominal cavity. The small intestine can then be inspected again for color and viability before the abdomen is closed.

If there is any question of viability of a segment, 100 per cent oxygen can be administered and the bowel observed.[1] If the color of the bowel definitely improves it should be viable.

If the ascending colon and transverse colon are seen in the right upper quadrant on opening the abdomen, the obstruction probably is due to a mesenteric band between the ascending colon and abdominal wall compressing the duodenum. This band should be cut and the cecum and ascending colon allowed to rest in the left side of the abdominal cavity with no attempt to rotate the colon to its normal position.

In older children and adults obstructing mesenteric bands are usually found in the region of the duodenum. (Figs. 6 and 7.) Occasionally a mobile cecum which has caused symptoms will be found lying in the right lower quadrant with an unattached mesentery. The peritoneal band between the hepatic flexure and the abdominal wall may be incised, permitting the right side

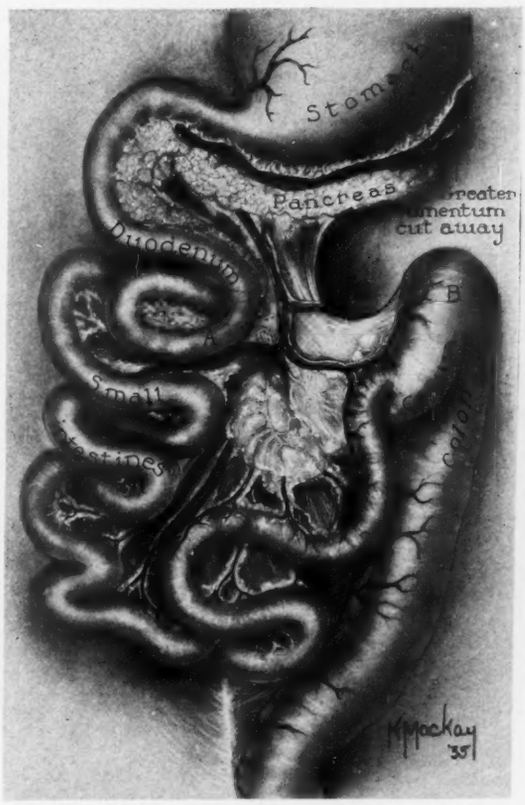

Fig. 9. Intestines of normal cat. This is the relative position of the human midgut before rotation begins. A–B is the duodenocolic isthmus, around which the midgut rotates. (From Wakefield, E. G. and Mayo, C. W. Intestinal obstruction produced by mesenteric bands, in association with failure of intestinal rotation. *Arch. Surg.*, 33: 47–67, 1936.)

of the colon to fall to the left side of the abdominal cavity. If the mesentery of the ascending colon is normally attached, the mobile cecum can be secured in the right lower quadrant with a few interrupted sutures.

CONGENITAL PERITONEAL DEFECTS

During the normal rotation of the midgut, the peritoneum of the mesentery fuses with posterior and lateral abdominal structures. Abnormal openings or rents may result from lack of complete fusion. Loops of bowel may pass through such an opening, forming an intra-abdominal hernia, which may become incarcerated. The most common site for a congenital rent is in the mesentery of the terminal ileum. (Fig. 13.) Occasionally there is a defect in the ascending mesocolon.

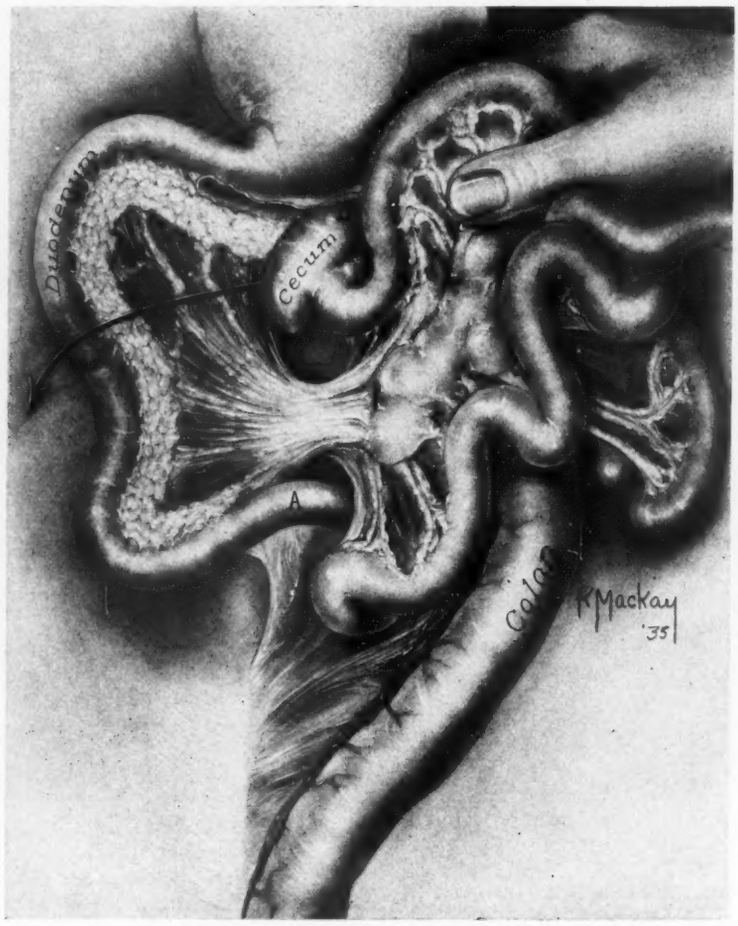

FIG. 10. Beginning of rotation. Duodenocolic isthmus (A-B) is now in the vertical axis. (From WAKEFIELD, E. G. and MAYO, C. W. Intestinal obstruction produced by mesenteric bands, in association with failure of intestinal rotation. *Arch. Surg.*, 33: 47–67, 1936.)

The symptoms are those of acute obstruction and surgical treatment should be instituted at once. Great care should be taken to avoid injury to the superior mesenteric vessels, which may course near the edge of the hernial ring and may be severed if an attempt is made to enlarge the ring to facilitate withdrawal of the bowel. Resection of a gangrenous segment of intestine may be necessary.

CONGENITAL ATRESIA OF THE INTESTINE

This uncommon congenital malformation occurs in two forms. The less common is a veil or diaphragm which completely obstructs the lumen. The more common consists of a thin threadlike remnant of intestine connecting the distended proximal bowel to the collapsed distal portion. This threadlike band connecting the two segments of bowel may be absent so that there is no vestigial connection between the proximal and distal segments or there may be several regions of atresia.

Atresia as well as stenosis occurs as a result of an arrest of fetal development of the intestinal tract during the second or third month of fetal life. There is a well-defined intestinal lumen at the fifth week of fetal life. Following this there is a rapid growth and proliferation of the epithelium of the intestine so that the lumen becomes smaller and obliterated and the intestine passes through a so-called solid stage. Cystic spaces or vacuoles then appear and coalesce, re-establishing the continuity of the intestinal lumen. (Fig. 14.)

FIG. 11. Rotation completed. Duodenocolic isthmus now reversed (B-A). Duodenum is now retroperitoneal and the mesenteric artery is on the right side of the vein. (From WAKEFIELD, E. G. and MAYO, C. W. Intestinal obstruction produced by mesenteric bands, in association with failure of intestinal rotation. *Arch. Surg.*, 33: 47–67, 1936.)

Diagnosis. Intestinal atresia should be suspected if progressive vomiting begins shortly after birth. Bile almost always is contained in the vomitus, as atresia is rare above the entrance of the common duct into the duodenum.

Farber[3] called attention to the fact that the amniotic fluid swallowed by the fetus contains epithelial cells from the vernix, which can be stained and identified from specimens of the meconium. In cases of atresia these cells, of course, are absent.

Roentgenologic examination reveals distended intestine or stomach above the lesion. Roentgenograms taken in the lateral position as well as with the infant held head down aid in determining the level of the atresia. Barium studies should not be necessary. They are dangerous and should not be attempted.

Treatment. Cases of successful treatment are rare but the mortality rate is 100 per cent if atresia is untreated; therefore surgical intervention should be attempted.

Parental administration of fluids, transfusion of 5 cc. of blood per pound of weight and an inlying gastric tube should be used preoperatively. If possible a side-to-side anastomosis should be made between the enlarged proximal segment and the small

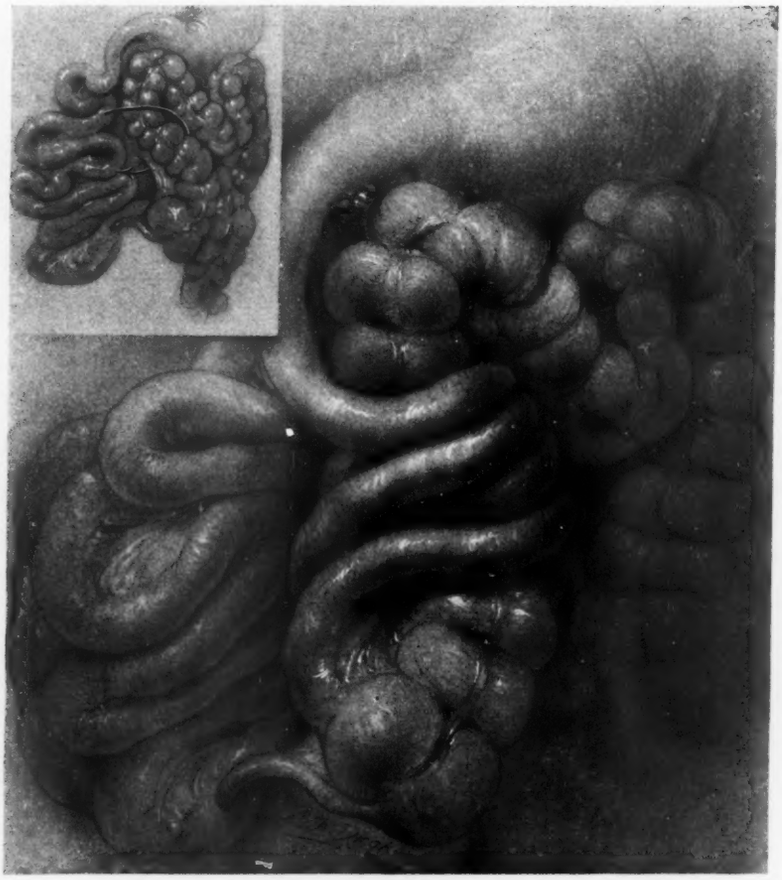

FIG. 12. Volvulus around a mobile right colon with malrotation of the midgut. Inset shows the colon on the left side of the abdomen and the intraperitoneal position of the duodenum. (From McKECHNIE, R. E., II and PRIESTLEY, J. T. Volvulus of small intestine: a report of thirty-seven cases. *Am. J. Surg.*, 34: 286–291, 1936.)

distal segment. This may be extremely difficult, but should be attempted even if the distal segment looks hopelessly small, as it soon acquires an adequate lumen. The distal segment may be distended with air as described by Webb and Wangensteen,[18] or the anastomosis can be made over a small catheter which is removed just before the anastomosis is completed, as described by Ladd.[9] This should be done with a single row of fine silk using a Connell stitch.

If the atresia is simply a diaphragm, it can be corrected by incising the bowel longitudinally, removing the membrane and suturing the bowel transversely, as described by Morton.[14]

CONGENITAL STENOSIS OF THE INTESTINE

Congenital stenosis in the small or large bowel is the result of the same type of arrest of embryonal development as is found in atresia, but the obstruction is not complete. Thus the narrowing may be of any degree.

The symptoms and physical findings vary with the degree and location of the narrowing. In eleven of twenty-two cases cited by Ladd and Gross the lesion was in the duodenum. Persistent vomiting usually occurs within a few hours after birth with loss of weight or failure to gain. Bile may be absent if the narrowing is above the papilla of Vater. Epithelial cells may be found in the meconium if the obstruction is incomplete.

In some cases of mild stenosis there may be vague digestive symptoms over a period of years. In one of the twenty-two cases reported by Ladd and Gross the patient was five years of age and another patient was nine years of age. Figure 15 shows a

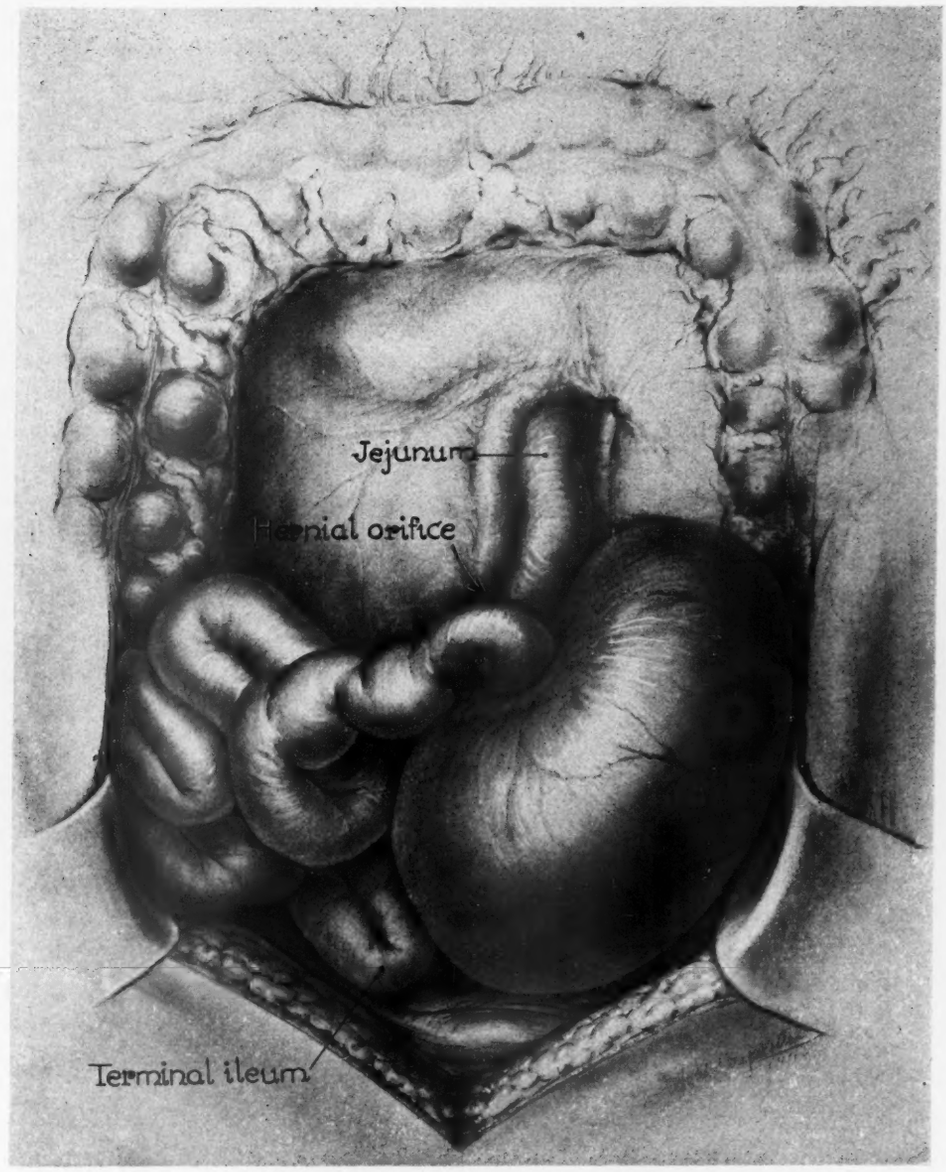

Fig. 13. Intra-abdominal hernia through a congenital defect in the mesentery of the terminal ileum.

congenital stenosis of the duodenum found at necropsy in a seventy-two year old man who had died of intestinal obstruction. He had had symptoms of intermittent obstruction since childhood. Barium studies may be helpful in questionable cases of longer duration, but barium must be given with caution.

Treatment. A short-circuiting primary anastomosis should be carried out. Dilatation of the stenosis with an instrument inserted into the lumen above or below the narrowing is unsatisfactory and should not be attempted. If the lesion is in the duodenum below the papilla of Vater side-to-side duodenojejunostomy should be done in preference to gastrojejunostomy.

CONGENITAL ATRESIA OF THE BILE DUCTS

Congenital atresia of the bile ducts is not an acute surgical condition but it will be reviewed briefly at this time. It was long thought that any attempt to repair congenital atresia of the bile ducts would be futile because of the associated cirrhosis and the involvement of the hepatic ducts in many cases. Following the work of

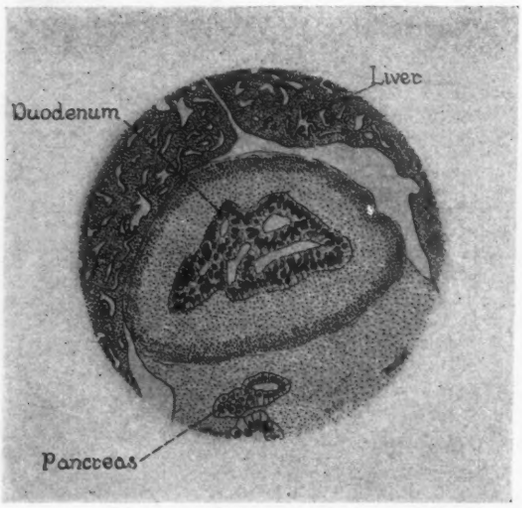

Fig. 14. Partial occlusion of the duodenum in a human embryo 18 mm. long. (From Nagel, G. W. Unusual conditions in the duodenum and their significance (membranous obstruction of the lumen, diverticula and carcinoma). *Arch. Surg.*, 11: 529–549, 1925.)

atresia was amenable to surgical reconstruction. As in the intestine, the lumina of the bile ducts are patent early in fetal life, then become filled with epithelium, then later again become patent.

Jaundice is the main symptom. This is usually present at birth but it may not appear for two or three weeks. Erythroblastosis foetalis and syphilis should be ruled out. Occasionally there is some tendency to bleeding and for this reason vitamin K should be given in all cases.

Clinically, the infant appears healthy and well nourished. There may be some difficulty with the formula because of poor absorption of fat, so it is well to feed a low fat formula until the time of operation. If sterile bile is obtainable from an adult patient with a T tube drainage, it can be given to the infant in the formula. There is

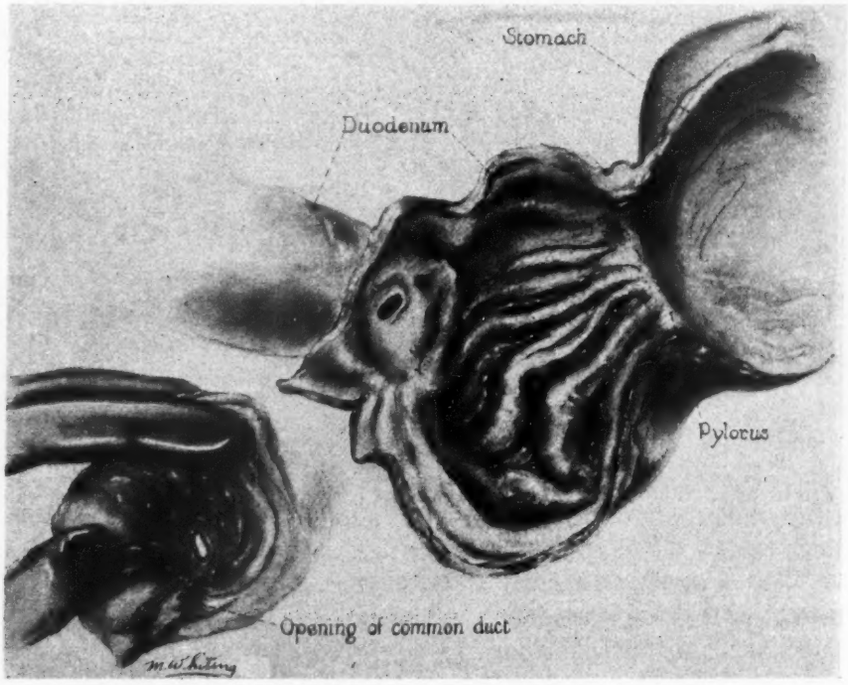

Fig. 15. Congenital duodenal stenosis in a man seventy-two years old. There had been symptoms of intermittent obstruction since childhood. (From Nagel, G. W. Unusual conditions in the duodenum and their significance (membranous obstruction of the lumen, diverticula and carcinoma). *Arch. Surg.*, 11: 529–549, 1925.)

Holmes,[8] who studied many examples of the various types of anomalies, it was realized that atresia of the bile ducts can be surgically corrected and is therefore worthy of exploration. In 37 per cent or fifteen of forty cases reported by Ladd[10] the no emergency about this condition and it is best to wait until the child is four or five weeks old before proceeding with surgical correction.

Atresia may exist in many forms from only fine fibrous cords representing the

extrabiliary ducts to atresia of only the distal portion of the common duct. If a portion of the common or hepatic duct is present, with or without a communication with the gallbladder, an attempt should be made to anastomose the duct with the duodenum with a single row of interrupted cotton sutures over a very small urethral catheter or vitallium tube. If a gallbladder is present and communicates with the common duct, it may be possible to mobilize the gallbladder from its bed and perform cholecystoduodenostomy. However, it is always better to make the anastomosis between the duodenum and the common or hepatic duct rather than the gallbladder even though the latter may be easier to do.

If a gallbladder is present it should first be opened to see if bile is present even though the ducts do not appear patent. Physiologic saline solution then can be injected and the ducts distended to determine their patency.

UMBILICAL EVENTRATION (OMPHALOCELE)

This congenital anomaly is due to the failure of part of the abdominal viscera to return to the abdominal cavity from the celomic cavity during the tenth week of fetal life. This failure is due to a disparity in size between the viscera and the abdominal cavity because of abnormally large intestines and liver, or retarded development of the abdominal wall.

The abdominal viscera at the site of the defect are covered only by a translucent layer of peritoneum and amniotic membrane. The umbilical cord is attached to the apex of the sac and the umbilical vein and arteries course over the membrane to enter the abdominal wall. The defect in the abdominal wall is usually 4 or 5 cm. in diameter, but may be considerably larger. (Fig. 16.) The membrane composing the sac loses its moist, pliable character after a day or two, becomes shriveled and dry and is easily ruptured.

Treatment. Regardless of how well an infant who has this anomaly may appear to be in the first few days of life, the infant will succumb to peritonitis if surgical repair is not instituted. As the membrane quickly drys out and there is no advantage in delay, operation should be performed in the first few hours after birth, before the intestinal tract becomes distended with food and gas.

FIG. 16. Umbilical eventration.

The skin surrounding the defect is incised close to the membrane. The umbilical vein and arteries are carefully observed and ligated below the skin level. An attempt should be made to dissect out the muscle and fascial layers so that the rectus muscles can be brought to the midline over the peritoneum and posterior fascia. If possible, the anterior fascia should be plicated with interrupted sutures of silk or cotton. If the abdominal cavity is too small to permit primary closure in layers, the skin and subcutaneous fascia should be undermined and then approximated over the intestine. Secondary closure in layers then can be done about a week later.

DIAPHRAGMATIC HERNIA

The thoracic and abdominal cavities, which freely communicate with each other in early fetal life, are later separated by the septum transversum developing ventrally and joining a mesodermal proliferation developing dorsally. Striated muscle develops between the layer of peritoneum and pleura, forming the diaphragm. This development is complicated by openings left in the posterolateral parts of the diaphragm, which later become closed. Congenital diaphragmatic hernias occur most frequently in this posterolateral region, the site of the embryonic pleuroperitoneal canal. A few hernias occur at the esophageal hiatus and still fewer in the retrosternal region. (Fig. 17.) Hernias occurring from an arrest of development early in fetal life when there was free communication between the two

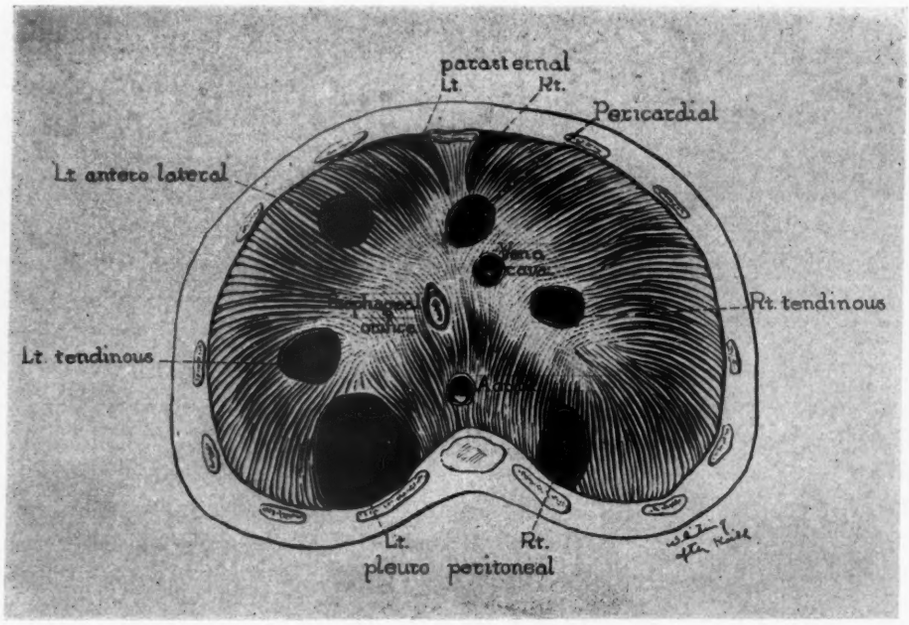

FIG. 17. Diaphragm seen from above, showing site of diaphragmatic hernias, both congenital and traumatic.

cavities will not have a sac. If the hernia has a sac, the arrest of development occurred after the peritoneal and pleural membrane separated the two cavities.

Diagnosis. Symptoms may be those of disturbed respiration, digestion or circulation. Dyspnea with or without cyanosis is common. It may be constant or may appear only when the infant is crying or feeding. The use of oxygen may be necessary. Examination of the thorax reveals decreased expansion on the affected side, a displaced apex beat, tympany or dullness depending on the underlying viscera and absent breath sounds. Vomiting may be the only symptom and occur with each attempt at feeding. The symptoms may be very mild and the diagnosis may not be made for several months. On the other hand, the symptoms may be those of circulatory failure and shock.

Roentgenologic examination should be done on any cyanotic or dyspneic infant as this usually immediately confirms the diagnosis. Barium studies should not be necessary and should not be used because of the danger of aspiration and subsequent pneumonia.

Treatment. As soon as the diagnosis is made, surgical repair should be instituted as these infants usually become worse with waiting and the risk increases. During the first day or two the gastrointestinal tract is free from gas and distention and the procedure is technically easier than it is later.

Fluids and oxygen should be given preoperatively and postoperatively and gastric suction should be instituted if there has been delay in diagnosis. Also an indwelling rectal tube may facilitate gentle squeezing of gas from a distended colon.

Positive pressure anesthesia may be necessary, as a communication between the pleural cavities may exist at the anterior mediastinum as demonstrated by Ochsner and his associates,[15] so that both lungs collapse when the pleural cavity is entered. Cyclopropane with its high oxygen content is desirable.

Phrenic crush immediately before the hernial repair is desirable as it facilitates the operation, and a high diaphragm affords more space in the abdominal cavity for the replaced viscera. In infants the abdominal approach should be used as there are no visceropleural adhesions and the viscera are easily pulled down from below. Reduction of the hernia by transthoracic approach would be impossible in many cases as the abdominal cavity is too small

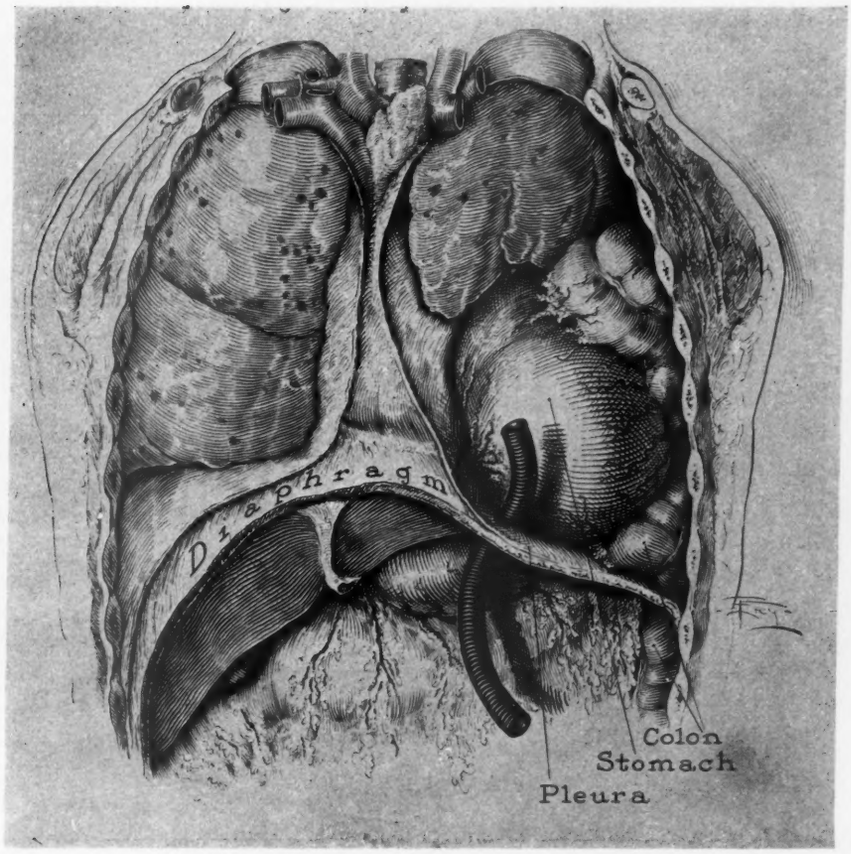

Fig. 18. Catheter inserted through the hernial ring so that air can enter the thorax and avoid development of vacuum as the viscera are withdrawn.

to hold all the viscera readily without causing respiratory or circulatory impairment.

Through a long left rectus incision, the muscle being retracted laterally to avoid injury to nerves, the viscera are withdrawn from the thorax onto the abdominal wall. This is done by first inserting a catheter through the hernial ring so that air can enter the thorax and avoid developing a vacuum as the viscera are withdrawn. (Fig. 18.) The viscera should be withdrawn systematically. In a left-sided hernia the stomach is withdrawn first, then the small intestine, the cecum, ascending colon and transverse colon and finally the spleen. In a right-sided hernia, the liver should not be withdrawn first even though this is the presenting organ. Such an attempt may damage the liver. The small intestine and then the colon should be withdrawn. The liver then can be brought down without difficulty.

The hernial ring should be closed with interrupted sutures of silk or cotton after the edges of the ring have been denuded. The abdominal viscera then are replaced into the abdominal cavity and the incision is closed in layers. Frequently this is difficult or impossible because of a small, underdeveloped abdominal cavity. If this is present, the skin and subcutaneous fascia are undermined and then approximated over the intestines. Within five to seven days the abdominal cavity will have stretched and a secondary closure in layers can be done before the cutaneous sutures cut through. Primary closure may increase the intra-abdominal pressure and cause death by respiratory embarrassment or circulatory collapse. If there is any doubt, primary closure in layers should not be attempted.

When a true hernial sac is found, a small hole should be made in it after the contents have been removed so that air may enter the pleural cavity and the sac may be with-

drawn. It is then excised and the hernial ring is closed.

Esophageal hiatus hernias comprise only a small group and seldom are acute surgical conditions. As the symptoms usually are mild, the diagnosis is often made when the child is several months or years old. Surgical repair is indicated only when the symptoms are marked. The esophagus may be congenitally short and barium studies will be necessary to determine this and to plan the surgical approach. A transthoracic approach should be used if the esophagus is to be freed up in an attempt to place the stomach completely in the abdominal cavity. If the esophagus is too short, the hernial ring may be sutured to the stomach to prevent the entrance of the other abdominal viscera.

CONCLUSION

In conclusion, the surgeon should be ever mindful that it is the function of nature to maintain life in childhood, and it is his purpose to aid nature in her efforts. When nature does fail in her efforts, or surgical intervention proves fruitless the physician should attempt to obtain permission for postmortem examination as it is through such study that progress is made in the treatment of congenital lesions.

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PERITONITIS*

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PERITONITIS kills either by an overwhelming toxemia or by the nutritional and chemical changes incident to the associated small intestinal obstruction. If persistent leakage from a hollow viscus does not occur and if the toxemia and the obstruction can be controlled, rarely should there be a death from peritonitis.

TYPES OF PERITONITIS

The first consideration in the treatment of peritonitis is whether or not a surgical operation is indicated. If there is progressive leakage from a perforated viscus, the patient will not survive unless the leakage is stopped. Within a few hours any free perforation of the gastrointestinal tract will give conclusive evidence of its presence by the appearance of gas in the peritoneal cavity. An *x*-ray in the upright or lateral position will establish the presence of such a perforation, and unless the perforation appears to be sealed off and the peritonitis localized or unless the leakage can be stopped (as by gastric suction in the case of a perforated peptic ulcer), the indications for closure of the perforation are clear.

Perforations of an appendix must be considered in a different category from other perforations of the gastrointestinal tract because in appendicitis there is almost never a progressive leakage of the contents of the bowel. The lumen of the appendix is so small that it becomes obliterated by edema and prevents soilage. At the same time protective ileus keeps the cecum empty. For these reasons the considerations which apply to other hollow viscera may be disregarded in connection with appendicitis. The problem in this case is the control of a localized but spreading infection by organisms which are apt to be both virulent and resistant to antibiotics.

Aside from appendicitis and perforations of viscera the causes of spontaneous peritonitis are few. Salpingitis, complications of pregnancy, and streptococcal or pneumococcal peritonitis in children are now relatively rare causes of serious peritonitis and are almost always caused by organisms susceptible to chemotherapeutic and antibiotic agents. Until localization occurs operation is no more effective than it would be in the treatment of a lymphangitis. In this type of infection penicillin is so effective that the problem of treatment is solved.

It is apparent that peritonitis groups itself into three great classes: The first is due to perforations of the gastrointestinal tract with continued leakage as seen in perforation of a carcinoma of the colon; the second is due to the spread of a mixed infection from a primary source such as the appendix; and the third is due to the rapidly spreading invasion of the peritoneum by organisms of high virulence but of marked susceptibility to chemotherapeutic and antibiotic agents. In postoperative peritonitis any or all of these types may be represented.

Spreading peritonitis following operation is rarely caused by mere contamination of the peritoneum with the contents of a normal gastrointestinal tract provided good aseptic technic is used. In order to establish peritonitis it is usually necessary either to have continued leakage or to have contamination with the virulent organisms from a diseased viscus. Minimal contamination from an abscess around a carcinoma or from a colon affected by ulcerative colitis is much more apt to cause peritonitis than

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extensive fecal soiling from a normal bowel, provided that continued leakage does not occur.

In the case of postoperative peritonitis it is necessary to decide whether the trouble is due to leakage or the result of contamination. The problem is rendered more difficult because after operation the presence of free air in the peritoneal cavity is to be expected and does not help to establish the diagnosis of perforation. Since the diagnosis is difficult to establish, and since a leaking anastomosis is difficult to repair, secondary operations, except for drainage, rarely are undertaken. One must then rely on antibiotics, gastrointestinal decompression, and general supportive measures.

FACTOR OF OBSTRUCTION IN PERITONITIS

Before the development of the Miller-Abbott tube, intestinal obstruction was perhaps the most important contributory cause of death from peritonitis. The abdominal distention with loss of large quantities of fluid and electrolytes into and from the gastrointestinal tract, the disturbances in nutrition and protein metabolism and in acid base equilibrium all were potent factors in affecting the unfavorable outcome. Today many of these factors can be prevented or controlled, and obstruction should be a rare cause of death.

The two most important considerations in passing a Miller-Abbott or Harris tube are the foresightedness of the attending physician and the intelligence, industry and perseverance of the house officer or nurse to whom the technical details are delegated. The percentage of cases in which a house officer can pass a Miller-Abbott tube is as good an index as any of his conscientiousness and intelligence.

Perhaps more important than the details of passing the tube is the time at which the decision is made to pass the tube. If it is delayed until the patient is distended, too ill to cooperate, and until the ileus is so marked that the bowel has lost its tone, it may be exceedingly difficult to pass. Under these circumstances it is best to put 3 or 4 cc. of mercury in the balloon or use a Harris single lumen mercury weighted tube. The patient is then taken to fluoroscopy where the tube usually can be passed promptly under visual direction. Once it is through the duodenum difficulties rarely are encountered.

If the tube is passed before operation in those cases in which the development of peritonitis or intestinal obstruction is considered likely, the entire procedure is facilitated. Similarly it is much easier to introduce the intestinal tube immediately after an operation such as appendectomy for a ruptured appendix than it is to try to pass it two days later when peritonitis is well established. If intestinal intubation is instituted successfully, the major problem of peritonitis is under control.

FLUID AND ELECTROLYTE BALANCE

Although volumes have been written on the subject of fluid and electrolytes in surgical patients, the problem is, in the main, a very simple one. If the plasma proteins and the cardiac reserve are normal, there is little danger of giving enough fluid to cause pulmonary edema or cardiac decompensation. If the urinary output is 1,500 cc. or more a day and if the patient does not have a chronic nephritis, there is little danger of giving too much salt unless all the fluids are given in saline. If the patient has a deficiency of chlorides, it is desirable to correct this as soon as possible; because if this deficiency is not corrected, the lowered chlorides will cause the breakdown of body protein and result in the accumulation of non-protein nitrogen in the blood.

If the renal function is good and if adequate salt and carbohydrate is given the kidneys and lungs will maintain normal acid base equilibrium. In elderly patients, however, and in those with damaged kidneys the precautions urged by Coller[1] should be followed lest the administration of excessive amounts of salt cause retention of salt with consequent retention of water and disturbance in the acid base equilib-

rium. In such cases ⅙ molar sodium lactate or plasma is preferable to salt.

As a working rule, the parenteral fluid intake should be 3,000 cc. of 5 per cent dextrose daily, 1,000 cc. of which should be in saline or ⅙ molar sodium lactate. To this is added the amount of salt and the amount of fluid lost by gastrointestinal intubation.

until penicillin became available in unlimited amounts that progress was made in controlling peritonitis due to mixed infections.

The results of prolonged treatment of a series of fifty cases of proved peritonitis of appendiceal origin with 100,000 units of penicillin, every two hours for four days followed by smaller doses for several more

TABLE I*

FLUID BALANCE FORM FOR TWENTY-FOUR HOUR PERIOD

Intake Mouth		Intake Parenteral		Output (Suction, Vomitus, etc.) cc.	Output Urine cc.
Type	Amount cc.	Type	Amount cc		
Water	500	5% glucose in saline	1000	1000	500
Water	500	5% glucose in water	1000	1000	500
Water	500	5% glucose in water	1000	500	500
		5% glucose in water	1000		
Total	1500	Total	4000	Total 2500	Total 1500
From Time 8:AM		Date 6/1/45		to Time 8:AM	Date 6/2/45

* Table reprinted from Hospital Care of the Surgical Patient by Crile and Shively, with permission of Charles C. Thomas.

As soon as the Miller-Abbott tube is passed there is no contraindication to the feeding of clear liquids by mouth. When the patient is taking fluid by mouth and has a tube in the stomach or intestinal tract, the problem of keeping an accurate record of the output from the tube and the oral intake arises. If the various types of oral and parenteral intakes and the urinary and suction outputs are not properly separated, the status of the fluid and salt balance cannot be evaluated. Table 1 affords a simple and effective means of recording these data in a form in which it can be visualized and calculated at a glance.

CONTROL OF THE INFECTION

There was no specific means of controlling pyogenic infections until the development of the sulfonamides and antibiotic agents. The sulfonamides were specific for infections caused by the hemolytic streptococcus or pneumococcus and helped to control the staphylococcus. But it was not

days has been reported in an earlier communication.[2] There was but one death in this series, and this did not occur as a direct result of peritonitis but from thrombosis of the mesenteric vessels. In all cases of spreading peritonitis the infection was controlled by penicillin. In all cases of localized peritonitis with or without formation of a mass, the infection was controlled and the mass eventually was absorbed. In no case was it necessary to drain an intra-abdominal abscess, and so far as is known there was no spontaneous drainage into the bowel.

The ability of Escherichia coli to destroy the activity of penicillin[2] could well account for the fact that massive doses of penicillin are required to control the gram-positive cocci when they are growing in a mixed infection associated with Escherichia coli. By giving enough penicillin over a long period of time eventually more penicillin is given than the Escherichia coli can destroy, and finally there is built up a

sufficient concentration of active penicillin to inhibit the growth of the virulent cocci.

Although it can now be seen how it is possible for large doses of penicillin given over a long period of time to inhibit the gram-positive cocci, it is still difficult to explain why penicillin, in concentrations insufficient to inhibit Escherichia coli, eventually results in control of infections in which this organism predominates. The most reasonable explanation is that Escherichia coli, in pure culture, is not a pathogenic organism but is in reality a mere saprophyte. If this were the case one need not rely on antibiotics to control it, as the normal defense mechanisms of the body would be able to deal with it once the gram-positive cocci were inhibited. The absence of cellulitis around the infected wounds of patients receiving large doses of penicillin, the absence of tenderness of large intra-abdominal masses, the absence of friability of the tissues overlying these masses, the absence of paralytic ileus in the face of diffuse peritonitis when adequate doses of penicillin have been given for a few days, all of these findings and the frequent observation that the temperature may be normal and the patient may feel well in the presence of a large intra-abdominal mass, point to the fact that it is not Escherichia coli but the gram-positive cocci that are the chief offenders in peritonitis. If the cocci are controlled, the peritonitis is controlled and the marvelous defensive and absorptive mechanism of the peritoneum is able to take care of the saprophytic or weakly pathogenic Escherichia coli infection.

In wounds, the defensive and absorptive action of the peritoneum is not available and, moreover, there is suture material, strangulated tissue, serum and blood in which Escherichia coli can grow. Even if it does not invade and cause cellulitis, a wound infection of low virulence is apt to develop. Instead of being absorbed, as it might be in the peritoneal cavity, eventually it drains. This explains the persistence of a normal expectancy of wound infections despite treatment with penicillin.

To check the *in vitro* resistance of mixed cultures to penicillin, a heavily seeded eighteen-hour agar slant of staphylococci Strain N.R. RL209 was washed with a six-hour broth culture of Escherichia coli. This was diluted to make a faintly turbid suspension and 0.01 cc. of this was used as the inoculum.

After twenty-four hours' incubation at 37°c. smears were made of all tubes and stained by Gram's method. (Table II.)

It is apparent that concentrations of penicillin of 0.039 unit per cc. inhibited the staphylococcus in pure culture, but that concentrations of 0.312 unit per cc.

TABLE II

COMPARISON OF CONCENTRATIONS OF PENICILLIN EFFECTIVE IN DESTROYING STAPHYLOCOCCI IN PURE CULTURE AS COMPARED WITH STAPHYLOCOCCI IN MIXED CULTURE WITH ESCHERICHIA COLI

Units of penicillin per cc. of medium.....	10	5	2.5	1.25	0.625	0.312	0.156	0.078	0.039	0.019	0.0097	0
Pure culture Staphylococcus aureus.....	−	−	−	−	−	−	−	−	−	+++	+++	++++
Mixed culture Staphylococcus aureus.....	−	−	−	−	−	−	+	+	++	++	+++	+++
Escherichia coli.....	+++	+++	+++	+++	+++	+++	+++	+++	++	++	+	+

− = no growth
+ = growth

were required to inhibit the staphylococcus in the presence of Escherichia coli. The bacteriology studies were conducted by Mr. Alfred Reich of Cleveland Clinic.

The above figures indicate that approximately eight times as much penicillin is required to control staphylococci in mixed culture with Escherichia coli as when the staphylococcus is in pure culture. This figure corresponds quite accurately with the increased dosage required to control mixed infections as compared with infections caused by gram-positive cocci in pure culture.

Escherichia coli in pure culture usually is of low virulence, and if the virulent gram-positive cocci are controlled by penicillin the peritoneum is able to localize and eventually absorb infections due to the remaining Escherichia coli.

Since the sulfonamides are ineffective against the colon bacillus in the concentrations which they attain by the systemic route, since they are ineffective in the presence of pus, since they do nothing that penicillin does not do better, and finally since they may prove toxic, there is nothing to be gained by their use in conjunction with penicillin in the treatment of peritonitis.

The bacterial flora involved in peritonitis of appendiceal origin are much the same as are present following perforation of a hollow viscus or in postoperative peritonitis, and the response of all these types of peritonitis to adequate doses of penicillin is much the same. The patients do not appear so sick after the first day of treatment. The peritonitis does not appear to spread after the first twenty-four hours. By the fourth day if progressive leakage has not occurred, definite clinical improvement nearly always takes place. The abdomen loses its exquisite tenderness after the second day, and if the intestinal tube is functioning there is less distention.

The temperature begins to fall on the second day of treatment and in most cases reaches normal in a week. The pulse rate remains slower than in uncontrolled peritonitis.

Pelvic or abdominal masses may appear during the course of treatment, but if 100,000 units of penicillin are given every two hours for from four to six days these usually resolve spontaneously and absorb without necessitating external drainage. Occasionally, a second course of treatment is required. A number of illustrative cases were presented in detail in a previous communication.[2]

ADJUNCTS TO TREATMENT

There are a number of helpful adjuncts to the treatment of peritonitis, most of which contribute to the patients' comfort but do little to alter the course of the disease. When the abdomen is distended, Fowler's position aids respiration but probably does little to localize the peritonitis. Hot stupes tend to relax the abdominal musculature and make the patient more comfortable but have not been proved to have any effect on the infection or intestinal motility. Drugs have little effect in controlling ileus resulting from peritonitis. Since an element of obstruction frequently is present, the benefits of a drug which increases peristalsis would be questionable even if such a drug were available.

Transfusion is of value when the patient is anemic and possibly when the serum proteins are low but otherwise does little for the patient with peritonitis. When edema develops it is usually a sign that too much salt has been given, and the most effective treatment is the withdrawal of salt and administration of water. If large amounts of blood or plasma are available, a transitory rise in serum proteins may be obtained, but both these and the amino acids have been disappointing in their inability to effect and maintain a significant rise in the serum proteins of a desperately ill patient.

MANAGEMENT OF INTRA-ABDOMINAL ABSCESSES

If peritonitis is treated early enough, long enough, and with adequate doses

of penicillin, the development of intra-abdominal masses which do not resolve after one or two courses of treatment is rare.

If a mass fails to resolve, there is rarely any hurry about surgical intervention. Penicillin usually controls the systemic reaction. A Miller-Abbott tube will obviate intestinal obstruction, and there is time to wait for the abscess to point so that it can be drained extraperitoneally. This is true of pelvic as well as abdominal abscesses, and here again it is best to wait until a fluctuant area can be felt by rectum or vagina. By this time the wall of the rectum is so friable that it is usually possible to penetrate it with the finger. In the case of the vagina it is more often necessary to make a small incision in the wall and insert a blunt instrument.

In some of the patients with peritonitis treated with penicillin, large pelvic masses developed and it appeared that they would point and drain. In two instances, fluctuant areas were palpable by rectal examination, but in neither did the wall of the bowel become sufficiently friable to allow introduction of the finger tip into the abscess. Both of these abscesses absorbed spontaneously without drainage. It was assumed that the penicillin controlled the cellulitis around the abscess to such an extent that the tissues remained healthy and prevented the pus from pointing.

SUMMARY

1. Peritonitis due to a ruptured viscus must be recognized early and the perforation closed.

2. Peritonitis due to contamination by a pure culture of a virulent organism is usually easily controlled by antibiotics or chemotherapy given in standard dosage.

3. Peritonitis arising from a mixed infection does not respond well to treatment with sulfonamides, and unless there is continued leakage, operation has little to offer.

4. If the toxemia and spread of peritonitis is controlled by massive doses of penicillin, if the obstruction is relieved by intestinal intubation, and if the fluids and electrolytes of the body are replenished the mortality rate of peritonitis should be low.

5. It is essential to intubate the small intestine and to give massive doses of penicillin as soon as the presence of peritonitis is recognized.

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EARLY INDICATIONS OF ADHESIVE SMALL BOWEL OBSTRUCTION*

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THE early diagnosis of small bowel obstruction is related to the recognition of three essential clinical manifestations of the disorder and to the proper interpretation of the radiographic findings of an *x*-ray film of the abdomen.

The clinical manifestations are those associated with a particular type of pain, visible peristalysis and borborygmus, which may be called the "Big Three" of small bowel obstruction.

The pain in this condition is quite characteristic and different from any other type of abdominal pain. It is not localized to any one quadrant of the abdomen but is general in type. It is not constant but periodic in character, lasts for a minute or so, is cramp-like in character, increases in intensity and ends abruptly, only to recur in a short time. It is not associated with any tenderness or rigidity except to a slight degree when the obstruction is of a strangulated type or there are involved adhesive bands to the anterior parietal peritoneum. Even then the tenderness and rigidity in no way approaches that associated with an acute localized inflammatory process or specific colic. In fact, the colic of bowel obstruction is unlike that of any other kind of abdominal colic because of its diffuse nature, intervals of freedom from pain and absence to a large extent of localized tenderness and rigidity.

Associated with this pain there is seen, moving across the abdomen in early obstruction, an intestinal peristalysis that is synchronous with the paroxysm of pain. This likewise is periodic in character and stops with the cessation of pain. Often these peristaltic waves may be seen to move across the abdomen in succession at the height of the crisis of pain. Later in the process, after distention has developed, the phenomenon may not be observed as intestinal distention lessens the violence of the contraction and the visibility of the peristaltic wave.

Correlated with the seizures of pain and the appearance of peristaltic waves there is heard upon listening to the abdomen a peculiar metallic sound which is synchronous with the pain and the visible peristalysis. The noise is unlike any other type of intra-abdominal sound, in that it is related in time, duration and termination, to the pain and peristalysis. The noise increases in intensity, has a metallic ring and ends abruptly in a crescendo of sound at the termination of the pain, only to recur with the next crisis of intestinal colic. This is well heard by auscultation with a stethoscope over the abdomen during a paroxysm of pain. It is known as borborygmus.

With a consciousness of the relationship of these symptoms and a recognition of their significance, a clinical diagnosis can be made of small bowel obstruction.

If there is a protruding mass on the surface of the abdomen significant of a strangulated bowel, the diagnosis is immediately confirmed; or if the abdomen shows evidence of one or more operative incisions, the suspicion of intestinal obstruction is further increased. These clinical manifestations associated with an increasing abdominal distention, without evidence of external strangulation or incisional scars upon the abdomen, likewise indicate bowel obstruction which immediately needs further confirmation.

The passage of gas, the movement of the bowels or adequate results obtained from an enema do not indicate that there is no

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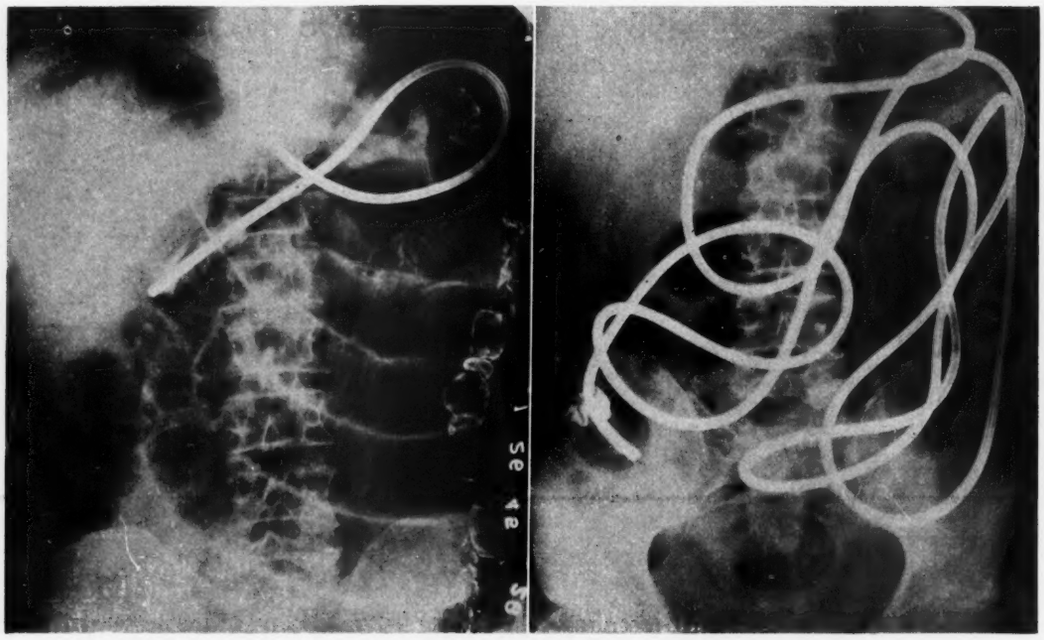

FIG. 1. Illustrates the pattern of bowel distention in simple, non-strangulating obstruction. The valvulae conniventes are clearly seen in this type of obstruction.

obstruction. It must be remembered that there is nothing wrong with the anatomy and physiology of the bowel distal to the obstruction and it can function adequately. This apparent function often gives a false feeling of security, only to find later a progressive obstruction with a possible peritonitis from a perforated strangulated gangrenous bowel. In fact, the distal bowel may empty itself from reflex action due to the presence of the obstruction.

This function of the bowel distal to the obstruction has been emphasized by Wangensteen and Goehl[1] and attention called to the disasters which may result if this is not fully recognized. Therefore, it can be said that the movement of the bowels, the passage of gas or adequate results from an enema by no means indicate the patency of the intestinal tract.

Regurgitant vomiting occurs early only in high intestinal obstruction. Since most small bowel obstructions are low, this type of vomiting does not occur until proximal bowel distention has become marked, abdominal distention is present and the clinical diagnosis is obvious. Early vomiting in low small bowel obstruction is reflex in character, as it is in colonic obstruction. Therefore, it can be said that vomiting in the absence of abdominal distention may or may not be due to obstruction and further investigation is necessary for diagnosis.

The *x*-ray is the only means by which a positive diagnosis can be made of early small bowel obstruction. By this means a diagnosis can be made, the approximate site of the lesion determined and the probable nature of the obstruction ascertained. In interpreting an *x*-ray film of the abdomen, it must be remembered that gas is normally visible in the stomach and colon but not in the small bowel except in infants. Gas is present but is not detectable by a radiographic film because it is mixed or emulsified with the liquid contents of the small bowel and becomes discernible only on an *x*-ray film when the continuity of the bowel is obstructed. Therefore, gas observed in the small bowel by this method is significant of obstruction and it can be detected in a few hours after it has occurred.

The pattern which the gas in an obstructed bowel assumes determines the probable nature of the obstruction. If the obstruction is of a simple type due to an angulation of the bowel from an adhesive band or a band passing across the bowel,

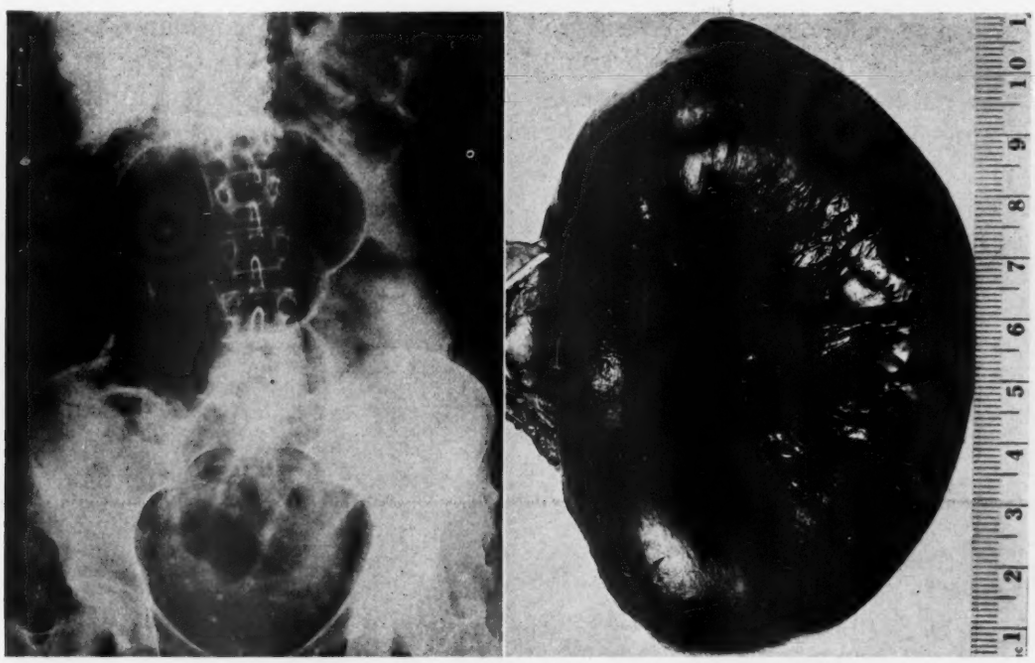

FIG. 2. Shows a distended loop of small bowel which does not assume any definite pattern of bowel distention, as is seen in Figure 1. The valvulae conniventes are not seen. The film is quite characteristic of strangulated obstruction. The resected specimen is shown.

the proximal portion becomes distended and in so doing gradually assumes a definite pattern in which the distended coils arrange themselves in a transverse position to the long axis of the body. The valvulae conniventes remain visible because the distended bowel is filled largely with gas and some liquid content and there is no extravasation of blood into the bowel, as is present in a strangulated obstruction.

This pattern becomes more pronounced with the advancement of the obstruction until ultimately a stairstep arrangement of the proximal distended bowel is observed. The thickness of the bowel wall indicates a relative degree of plastic exudate, fluid or peritonitis.

A strangulated obstruction, however, presents no such arrangement of the bowels in this transverse position. There is no definite pattern assumed by the distended coils of the bowel in this type of obstruction. The distended loops arrange themselves in whatever portion of the abdomen the obstruction occurs and no characteristic pattern is assumed and proximal distention above the site of strangulation is slow to develop, as emphasized by Wangensteen. In contrast to a simple non-strangulating obstruction, the valvulae conniventes are not seen or easily detected due to the extravasation of blood into the strangulated loop of bowel and into the free abdominal cavity.

It is important to differentiate, if possible, between a simple non-strangulated obstruction and a strangulated one. In the former the visibility of the bowel is not in danger and operation can be deferred until the patient can be physiologically prepared for operation by chemical and fluid rehabilitation and the distended bowel decompressed by intestinal intubation. In strangulated obstruction the blood supply is damaged, bowel gangrene and perforation are imminent and operation cannot be deferred. Physiological rehabilitation must be deferred until during and after surgery because of this impending danger.

Through careful correlation of the radiographic pattern of the distended bowel with the findings at surgical exploration we feel quite definitely that a competent radiologist can be of great assistance in determining the presence of small bowel obstruction, the approximate site of the lesion and the

probable nature of the disorder. We have found that by a careful study of the pattern of the gaseous distention the immediate urgency of the situation can be determined. We believe, always, in the immediate operation of all early small bowel obstructions, but there are many with marked fluid loss and great abdominal distention, who are not good subjects physiologically or anatomically for immediate surgery. If in these cases a careful study of the pattern of gaseous distention, as observed in a radiographic film, the lesion appears to be non-strangulating, physiological rehabilitation and intestinal intubation may be safely accomplished prior to surgery. The operation is then one of election and is much more satisfactorily and safely done, than when great distention is present and anatomical difficulties are great and hazardous.

Figure 1 demonstrates the value of the *x*-ray in evaluating the type of small bowel obstruction. It shows the transverse pattern of the bowel distention and the valvulae conniventes are clearly seen. The clinical manifestations and radiographic manifestations are those of simple obstruction with no evidence of vascular damage. The Miller-Abbott tube decompressed the distended intestines and aided materially in the surgical approach. Operation revealed a simple adhesive band obstructing the small bowel by constricting the lumen. Recovery was prompt after freeing the constricting adhesion.

Figure 2 shows an irregular type of small bowel distention that conforms to no pattern that is related to the long axis of the body and the valvulae conniventes are not seen. The clinical manifestation shows an uneven distention in the contour of the abdomen with slight localized tenderness and rigidity. Immediate operation disclosed a small bowel loop twisted upon itself by a band of adhesions attached to the bowel mesentery. The bowel was black and required resection. Recovery was uneventful after resection and anastomosis. The *x*-ray was valuable in determining the nature of the obstruction and the need for immediate surgery.

We have found the correlation between the *x*-ray diagnosis by means of a flat film of the abdomen and the operative findings to be very accurate and have previously reported this relationship in August, 1944.[2]

We, therefore, emphasize the importance of competent radiological assistance in evaluating the nature of the obstruction of the small bowel and the need for immediate surgery. We know that in some instances of advanced simple obstruction surgery is more safely and effectively done after physiological rehabilitation and intestinal intubation by the Miller-Abbott tube has been satisfactorily employed.

SUMMARY

The clinical symptoms and signs of small bowel obstruction are characteristic and are unlike any type of intra-abdominal disease. The colic is specific in character and different from other forms of abdominal colic. The *x*-ray is most valuable in confirming the diagnosis of small bowel obstruction, in locating the site of the obstruction and in determining to a large extent the nature of the obstruction.

The function of the bowel distal to the obstruction is unimpaired and should adequate function take place it does not indicate the absence of obstruction.

The urgency of immediate surgery is emphasized in strangulated obstruction in contrast to the feasibility of delaying surgery in advanced simple obstruction until physiological measures and intubation procedures can be adequately employed.

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GUNSHOT

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GUNSHOT in warfare provides an abundance of casualties having every conceivable type of injury. Blast and burns, which may accompany highly explosive ordnance with metallic fragments of every known composition and size, complicate the medical care of these patients. The treatment of such wounded requires a fund of knowledge of the basic sciences with the exercise of immediate good judgment. In addition, there is needed the requisite technical skill of surgical procedures with an added ability to improvise.

No less can be said for the medical care of gunshot in civil practice except that the casualties are sporadic and fewer in number, and the missiles usually conform to specifications of size and composition. Blast and burns rarely complicate civilian gunshot, and there are usually available immediate adequate facilities and skill for their care.

We are reasonably certain that the removal of accessible bullets and foreign bodies was of paramount importance in all previous wars. This notion of mandatory removal persisted among many of us for a short time early in World War II, until the multiplicity of various sized fragments, widely scattered, made their removal utterly impossible. We soon adopted the principle of removing only those foreign bodies easily approached without producing permanent or extended disability, and in addition, those which caused interference with vital, visceral or skeletal functions.

Especially those foreign bodies lying within the walls of the torso or in the extremities, with few if any symptoms and not complicated by a hematoma, clothing and earth, were allowed to remain and to be removed at a much later time, and then only if deemed necessary. Possible accompanying anaerobic infections received minor consideration because in this past war there was a well defined program of prophylaxis. Non-removal of inocuous metallic foreign bodies might well be adopted in civilian gunshot except for the legal aspects occasioned by the need for identification and ballistics. Since civilians are not prophylactically prepared against tetanus, gas gangrene and other infections in gunshot, the elements of drainage, inoculations, and chemotherapy must assume real importance.

Early in the war, foreign metallic bodies deeply embedded in the substance of such solid organs as the liver presented the problem of necessary removal. No former experience of anyone, nor available medical literature, could definitely provide us with the required information. Uncertainty prevailed as to whether the surrounding zone of necrosis seen in the roentgenograms might not lead to progressive parenchymal destruction or perhaps to the formation of an abscess or cystic cavity. Furthermore, in the surgical removal of deeply embedded bullets and small to moderate sized fragments from the liver, there existed the necessary trauma incident to localization and extraction with a fair possibility of not readily finding the missile. Finally, it was decided that such foreign bodies should be left in the liver and that this injured viscus would recover and encapsulate the object, without appreciable parenchymatous damage. Accordingly, several such patients harboring foreign bodies in the liver were closely observed clinically for several weeks with *x*-ray studies at intervals. The initial zone of surrounding necrosis disappeared after three or four weeks and the patients apparently suffered no ill effects. In order to verify our findings and opinion, we aseptically implanted previously removed metallic fragments into

Fig. 1. Photomicrograph showing foreign body scar tissue in upper one-half, and normal liver cells in lower one-half. The scar tissue is encircling a strand of liver cells.

the liver of several rabbits. These animals were carefully observed clinically and studied roentgenologically at various times for a period of two years. They were then sacrificed, and at autopsy there was found in each instance a dense fibrous capsule about each foreign body, with no apparent damage to the parenchyma surrounding this fibrous capsule. (Figs. 1, 2 and 3.) Discussions with others having the same experiences led us to conclude that the liver entertains metallic fragments exceptionally well and in the manner as does other living elements supplied with reparative connective tissue.

Gunshot of the abdomen is cause for urgent and judicious care. Besides the shock, there is concealed trauma consisting usually of perforation and laceration of viscera and blood vessels which give rise to various degrees of hemorrhage and infection. The difficulty of knowing the extent of the injuries, the optimum time to explore, and where the incision is to be placed, arise immediately and must be considered while the patient is undergoing the treatment for shock.

These considerations become manifest after a close inspection of the position and character of trauma at the points of missile entrance and exit, and the line of missile travel which indicates the viscera likely injured. From these observations, one learns whether the track is through and through or tangential, and in addition, deduction is made relative to the amount of trauma caused by the wobble of the bullet or fragment. In many instances, a retained missile located by an *x*-ray exposure at a distant area from the point of entrance complicates the assessment of the damage likely present. We recall one instance of a Japanese prisoner of war having a three-weeks' abdominal gunshot suddenly presenting the findings of a very acute appendicitis. An *x*-ray revealed a bullet to be retroperitoneal and under the cecum. A wholly different and possibly more successful surgical procedure was of course adopted.

The management of the severe shock usually present must have the first consideration. We found that ½ to ⅓ gr. of morphine administered intravenously, seemingly a forgotten procedure in civil practice, was found advantageous for immediate sedation. The utmost care was and must always be exercised to prevent dangerous morphinization, as varying doses of the drug may have been previously ad-

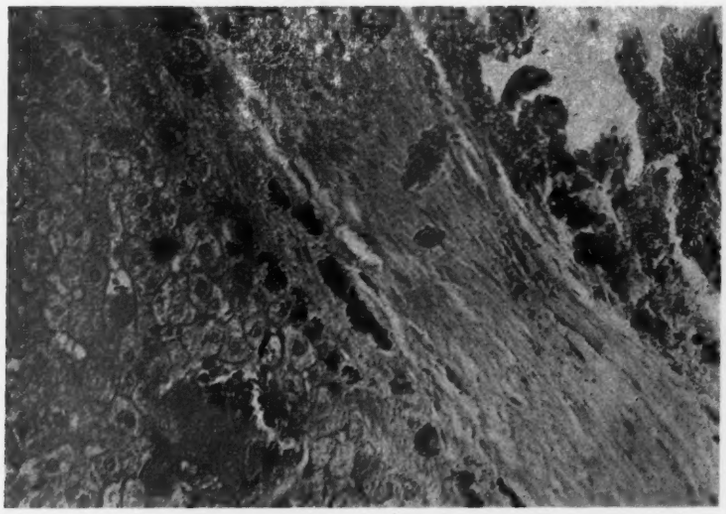

FIG. 2. A high power photomicrograph showing the sharply defined connective tissue enveloping a metallic fragment with surrounding healthy parenchymal liver cells.

ministered and unrecorded. Sufficient amounts of intravenous fluids, consisting of whole blood or plasma, with or without glucose-saline and serum albumin, if available, must be given immediately. We were often compelled to explore a patient still in shock, the relief of which required immediate and secure ligation of bleeding blood vessels or the placement of retaining sutures to stay hemorrhage of a solid organ. Serum albumin (Cohn), as previously mentioned, was found to be a very valuable agent, as it increases the blood volume by reverse osmosis in an amazing manner. On occasion when immediate venoclysis was necessary, we adopted the procedure of inserting the needle into the femoral vein in the groin, thus sparing the time necessary to expose surgically a collapsed vein. This allowed for immediate, rapid and copious intravenous therapy. We are certain that femoral vein puncture, another often forgotten procedure, can be utilized in civilian practice, especially in instances of severe injury complicated by extensive body burns.

In our opinion, the optimum time for surgical intervention, excepting those requiring immediate control of hemorrhage, averaged about two hours from the time of injury.

The incisions used and found most appropriate were the right or left paramedian, so placed to afford ready access to the intra-abdominal injury. At times the entrance defect caused by missile was enlarged after a careful wound toilet with excision of the ragged wound edges. A second appropriately placed incision was often very valuable in facilitating further mobilization to expedite the surgery. On occasion assistants were delegated to make repairs wholly separate from the lesion immediately being cared for by the surgeon. Transverse incisions, although at times appropriate for good exposure of the upper abdominal injury, were rarely used because they result in weak scars and postoperative hernias, especially so if drainage must be instituted.

After exposure and cleansing of the peritoneal cavity, hemorrhage requires primary attention. Brisk bleeding arises usually from such solid organs as the liver, spleen, kidney and pancreas, and most often from lacerated mesenteric blood vessels. Following secure ligation of the blood vessels, the injured solid organs must be repaired. Large bleeding defects often require only vascular ties and sutures. Occasionally, a defect must be packed, using if possible some available omentum. Several instances required sponge packs deeply buried in the parenchyma of an

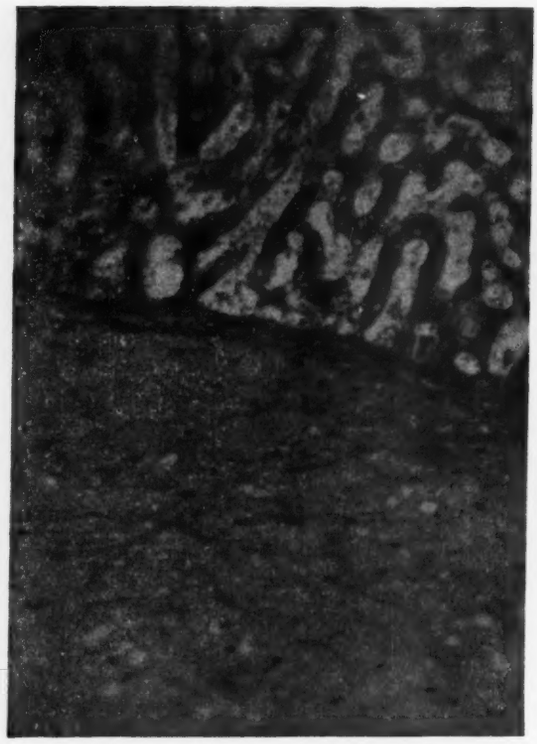

Fig. 3. A much higher power photomicrograph revealing the sharp outline between strands of healthy rabbit liver cells and the underlying dense fibrous foreign body capsule.

organ, such as the liver, with closure of the abdomen and removal at a later well selected time. Without exception these patients requiring such heroic measures did not survive. Fibrin foam[1] was found to be a valuable agent to assist in the stay of moderate parenchymatous hemorrhage. This most valuable agent should be made immediately available to all hospitals. Oxidized cellulose,[2,3] used as absorbable dry sponge packs, is now available and should be found to be very efficient for staying major parenchymatous hemorrhage. The necessity for the dangerous removal of ordinary non-absorbable gauze packing can be eliminated since this oxidized cellulose is completely absorbed in a few days. We have had no experience with its use, but we can envision the usefulness it might have provided in many fatal instances.

Peritonitis due to perforations of the alimentary tract must be the next important consideration. Lacerations may require only simple suture, but often one or more resections may be necessary. A careful examination is always made to prevent leaving behind a small unrecognized lethal puncture or laceration. When resections are necessary, we found that end-to-end anastomosis with a technic to prevent stenosis, using non-absorbable reinforcing sutures, was expeditous and rewarded us with excellent results. Following repair of the intra-abdominal injury of any type, we unhesitatingly irrigated the abdominal cavity, using copious quantities of warm saline solution with suction. We are convinced that this procedure is valuable because it removes the free blood, débris and contaminations, and in addition one obtains a more precise view of the repaired damage. In addition, 5 to 10 Gm. of sulfanilamide were usually sprinkled into the peritoneal cavity. On many occasions 100,000 units of penicillin were also instilled through a catheter which was withdrawn when the incision was repaired. The danger of peritoneal chemotherapy is that an occasional patient may suffer a severe reaction by being sensitive to such drugs. This was encountered several times, especially in sulfa sensitive individuals. This should have thoughtful consideration and be of great concern to surgeons in civil practice where one must deal with patients having had these agents used promiscuously for trivial ailments. After closure of the abdomen and with dependent and preferably suction drainage when necessary, we always added internal drainage by using a Miller-Abbott tube loaded with 6 cc. of metallic mercury and often passed by direct fluoroscopic vision. This manipulation under the fluoroscope assists in an accurate and rapid transit of the bulb to the pylorus, facilitating duodenal entrance in a short period of time. In several instances this tube was usually guided into the duodenum by manual manipulation when the operation was completed and the abdomen was still open.

A retroperitoneal hemorrhage or hematoma always requires careful investigation since the retroperitoneal organs, such as

the kidney or a fixed portion of the colon, may be seriously injured. A severely damaged kidney can usually be drained and left for a later and more considerate treatment. The same is not true relative to retroperitoneal colon perforations, for if left unrecognized and untreated, serious spreading retroperitoneal infections and abscess formations occur, enhanced, of course, by a good culture medium.

Other colon injuries localized by search and smell require skill and ingenuity in their surgical management. Simple suture of a colon defect, however well done, is rarely adequate. Injured sections are usually treated by exteriorization. When repaired *in situ*, a supplemental colostomy or other adequate vent for gas pressure and fecal stream becomes a most judicious procedure. We were able in a few instances to insert a large calibre mushroom retention catheter through colonic perforations. It was our experience that when this catheter was strung through omentum and out through a small stab wound and removed on the seventh to tenth day, the fistula closed promptly. Posterior retroperitoneal perforations were treated by catheter in the same manner and surprisingly without any extended invasive retroperitoneal inflammation. Perhaps efficient dependent and suction drainage with chemotherapy administered at the time of operation and during the postoperative period provided this safety. The same general principles for the postoperative care of patients with colon surgery were the same as were provided for those with injuries of the small bowel. Adequate but not excessive fluids with sufficient amounts of protein, carbohydrate and vitamins were carefully maintained.

Injuries to the spleen, when minor and not confined to the pedicle blood vessels, including small through-and-through bullet tracks, are usually not bleeding when exposed. We are certain that many spleens, having minor injuries which would heal spontaneously, are unnecessarily removed. In support of this statement, we had occasion to observe at interval inspections, by means of the periteoneoscope, the rapid and complete healing of minor splenic lacerations.

Gunshot of the pancreas is a lesion that must be recognized. Such an injury requires good exposure and a very careful survey of position and extent. Carefully placed non-absorbable sutures that do not ligate or stenose the main ducts in the parenchyma, but control the hemorrhage, may promote survival of one having this dangerous lesion. Absorbable catgut sutures are less valuable because they are quickly digested. One patient having a pancreas almost severed by a crush against the spine survived under this management. Appropriate dependent or preferably suction drainage seems a prime necessity to prevent wide spread of the autolytic enzymes. The omentum was, in several instances, successfully utilized as a funnel embracing wicks which served efficiently as a barrier to the spread of pancreatic juice.

Thoraco-abdominal gunshot usually provide the most serious problem. Usually the liver, spleen, diaphragm and lung are injured. On most occasions operative repair was made through the original thoracic wound enlarged sufficiently to allow good vision and mobilization. The rent in the left diaphragm can be enlarged to perform splenectomy when necessary. After repair of the diaphragm and the thoracic incision with good postoperative management, including chemotherapy, these patients not too seriously injured survived. We can recall no instance of survival of such an injured man in whom the wobbling missile of any size traversed the liver from side to side with an explosive exit, and with injuries extensive enough to require parenchymal packing with gauze.

A skilled anesthetist is an important member of the surgical team. The repair of abdominal gunshot requires relaxation for adequate exposure. Spinal anesthesia, often continuous, is the anesthesia of choice. Occasionally, in prolonged procedures it must be supplemented with intra-

venous pentathol and quite often with curare. This requires experience and judgment in knowing when and how to use these agents. Gas inhalants with positive and negative pressures must often be used, especially when the thorax is involved.

A statistical analysis is shown (Table 1) of the civilian gunshot in Los Angeles during the years 1940 to 1945, from the records of the Los Angeles General Hospital:

TABLE 1

Year	Total Cases	Operated	Died	Post-operative Death
GUNSHOT WOUNDS OF THE ABDOMEN				
1940–41	29	21	10	6
1941–42	35	25	10	8
1942–43	36	25	12	8
1943–44	46	31	7	3
1944–45	31	21	10	7
	177	123 (60%)	49 (27%)	32 (26%)
GUNSHOT WOUNDS OF THE CHEST				
1940–41	33	3	6	1
1941–42	43	4	3	2
1942–43	32	7	10	3
1943–44	30	1	2	..
1944–45	35	2	6	1
	173	17 (10%)	27 (15.6%)	7 (41%)

This tabulation of the civilian gunshot patients treated at the Los Angeles General Hospital, during the years 1940 to 1945 reveal a total of 350 patients. Abdominal and thoracic gunshot are about equal in number. The records reveal that 60 per cent of those with abdominal gunshot were subjected to operation with a 26 per cent mortality. There was a 10 per cent mortality on non-operative cases.

Of those with thoracic gunshot only about 10 per cent were subjected to operation, with a mortality of 41 per cent. The mortality of all thoracic gunshot was 15.6 per cent.

A study of the combination of both abdominal and thoracic gunshot in the same individual, with surgical procedures and causes of death, cannot be within the scope of this paper. This chart merely serves to indicate roughly the statistics of civilian gunshot from a general hospital in a large city. A detailed study of all hospitalized gunshot in this city, with the details of the medical care, would be of great interest to me and perhaps to others who have recently cared for victims of gunshot in war.

SUMMARY

This paper is based on four years of active service as a surgeon in the past war. It seems appropriate to discuss and perhaps apply some of the principles of management of gunshot in war to civilian gunshot which will regrettably remain an occasional part of surgical practice. Active prophylaxis against anaerobic infection in our war gunshot is such a strict program that there remained the rare necessity of removal of most foreign bodies and even drainage of wounds. The same may not be true in civilian gunshot except when prophylaxis is immediate and efficient. Metallic foreign bodies are well tolerated in the liver as shown by clinical and experimental studies.

Survivors of thoracic gunshot are seldom subjected to surgical repair, while abdominal gunshot victims are seldom denied celiotomy. The relief of shock and stay of intra-abdominal hemorrhage with repair of lacerations is paramount. External and internal drainage with suction and an active chemotherapy seem important. Blood and plasma, and in addition, fluids containing an appropriate amount of proteins, carbohydrates and vitamins given intravenously are essential in the postoperative regimen.

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THE PRESENT STATUS OF THE PROBLEM OF REGIONAL ILEITIS*

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SINCE the classical paper on Regional Ileitis by Crohn, Ginzburg and Oppenheimer published almost fifteen years ago, a great amount of material has accumulated and certain definite opinions have been formed which deserve repeated emphasis. In talking to physicians from various parts of the country, it becomes apparent that, in spite of repeated publications on the subject, there is still considerable haziness concerning the clinical features of the disease and far more bewilderment as to what treatment constitutes the most desirable and effective method of therapy. It is the purpose of this short paper to relate our experiences with this disease in the hope that many of these debatable points may be clarified.

Our experience to date is concerned with the management of approximately 200 instances of this disease. Careful appraisal of the clinical histories of these patients has led to the grouping of symptoms into five main categories:

Acute Ileitis. There is usually no preceding history and the onset of the disease is fairly sudden, closely simulating that of acute appendicitis. In fact, in every instance of acute ileitis, operation was undertaken under the mistaken diagnosis of acute appendicitis. It is very doubtful if the differential diagnosis can be made preoperatively, except in rare instances.

Symptoms of Enteritis. The triad of foul non-bloody diarrhea and cramps, loss of weight and anemia is fairly constant in this group. Fever may or may not be present, its occurrence depending a great deal on the extent of disease outside the bowel wall. It must be remembered that ileitis, so far of unknown etiology, starts as a mucosal ulceration, usually on the mesenteric side of the bowel, progresses slowly, is accompanied by thickening of all layers of the ileum and is peculiarly characterized by perforation of these ulcers toward the mesentery with frequent formation of mesenteric abscesses. The latter is frequently the most common cause for the fever. As the inflammation progresses slowly, the diseased segment may become attached to the abdominal wall or to adjacent viscera, such as the cecum, small bowel, sigmoid, bladder, pelvic organs or ureter. Continuation of the infection may result in the spontaneous perforation of one of these abscesses or ileal ulcers through the abdominal wall or one of the hollow viscera. The *non-bloody* diarrhea is one of the main distinguishing clinical features from non-specific ulcerative colitis. This should be emphasized repeatedly. Another frequent clinical finding is the presence of perianal abscess or fistulas. These are due to crypt abscesses developing on the basis of the foul diarrhea, rather than to direct fistulous communication between the diseased ileum and the pelvic floor.

Symptoms of Obstruction. In a small group of patients, the disease remains confined to the ileum, progressing to a stage of great thickening of the bowel wall due to deposition of the products of inflammation, and marked diminution in the caliber of the lumen. Under such circumstances, symptoms of small bowel obstruction supervene, frequently clouding the true underlying process. I have seen five such cases and, in one, the symptoms of acute intestinal obstruction were evident on admission to the hospital. Only careful inquiry into the previous gastrointestinal history will lead to an accurate interpretation of the underlying pathological process.

Symptoms Caused by Involvement of Adjacent Viscera. As already indicated, the

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diseased segment of ileum may become adherent to a neighboring viscus and produce symptoms which might be interpreted as originating from primary disease of this viscus. Thus, fixation to the bladder will result in symptoms mainly of a urological character. The development of a spontaneous fistula into the bladder leads to the passage of gas, and possibly fecal material, per urethra. The occurrence of such symptoms in a young individual (and ileitis is by and large a disease of the young) should lead the physician to suspect ileitis as the underlying pathological process. I have seen four instances of enterovesical fistulas caused by regional ileitis. Fixation of the diseased loop in the female pelvis may lead to bizarre gynecological symptoms and physical signs. Finally, spontaneous perforation may occur into adherent cecum or sigmoid. These may be demonstrable on *x*-ray examination.

Presence of External Fecal Fistulas. There is considerable misinterpretation of the exact pathogenesis of these fistulas. The belief seems to be widely held that they are due to the "blowing out" of the stump following appendectomy which is frequently performed under a mistaken diagnosis. However, the essential factor is the perforation of one of the ileal ulcers into the mesentery with the formation of an abscess and fixation of the abscess to the abdominal wall, followed either by operation or spontaneous opening externally on the abdominal wall, usually in the right lower quadrant. In my experience, the most common cause for a fecal fistula following appendectomy has been an underlying ileitis. These fistulas may be multiple. In one instance, twenty-six such fistulas were counted on the abdominal wall. Because of the origin of the fistulas from the diseased segment of bowel, the fistulous tract from the bowel to the skin surface may be long and tortuous. This point should be remembered when we discuss treatment.

The examination of a patient suspected of harboring regional ileitis may disclose interesting physical signs. Usually weight loss is obvious and anemia of mild degree is present. One may palpate an ill-defined mass in the right lower quadrant which may become more clearly delineated on bimanual or rectal examination because of the well known tendency of the terminal ileum to lie downwards towards the pelvis. The presence of one or more fecal fistulas on the abdominal wall will aid in corroborating the clinical impression gained from the history. One must never fail to complete the examination by inspection of the anal region for fistulas and by a careful digital exploration of the rectum. I wish to emphasize the need for sigmoidoscopic examination to exclude the presence of ulcerative colitis.

What more can the physician do to establish definitely the diagnosis of ileitis? An *x*-ray examination of the colon by barium enema should be done. This will accomplish two purposes: firstly, it will exclude the presence of a concomitant ulcerative colitis; secondly, it may demonstrate the narrowed diseased ileum by passage of the barium through an incompetent ileocecal valve. Whether the disease is demonstrated by barium enema or not, it is important to supplement this examination by a regular gastrointestinal series by mouth. I wish to emphasize particularly the need for hourly or two hourly observations of the small bowel during this examination because it is important to demonstrate the presence or absence of diseased segments in the jejunum or upper ileum. The so-called "skip areas" are a characteristic feature of ileitis and they may be demonstrated in the *x*-ray films. However, it is more important for the surgeon to look for them at the operating table. The characteristic finding in ileitis is the so-called "string sign," described by Kantor. It is produced by the narrowing of the lumen in the diseased bowel because of the thickening of its wall. This typical *x*-ray finding is pathognomonic of regional ileitis.

On the basis of a large experience to date, we at Mount Sinai Hospital, are convinced that ileitis should be considered a surgical disease and treated as such. It is possible

that, with the disclosure of its exact cause, we may change this opinion; but at the present writing our best therapeutic results have followed surgical therapy. Occasionally, one sees a patient with a short history of mild diarrhea and fairly suggestive *x*-ray findings who maintains his weight, has no disabling symptoms and seems well in all other respects. Should such a patient be subjected to operation? We have advised against operation in such instances and have followed the patients for varying periods of time. The majority of these patients have remained relatively well and there has been no change in the radiographic findings. It seems to me that the indication for surgical intervention in such cases should rest with the severity of the symptoms.

I have been asked on many occasions what should be done when the surgeon finds an acute ileitis during the course of an operation for supposed acute appendicitis and the opinion has been expressed that, if an acute ileitis without perforation is found, nothing should be done. It is questionable if even appendectomy is advisable. The reason for this opinion is twofold: First, the patient usually is not prepared for an intestinal anastomosis, but what is more important, it is impossible to predict what the outcome in such cases will be. In the majority of such cases in our series, there have been no further symptoms and the patients have remained well, some for as long as fourteen years. In three instances, recurrence of symptoms necessitated operative intervention. We have been impressed with the tendency for spontaneous remission not only in the acute cases, but in many patients with extensive involvement of ileum and jejunum which are not amenable to surgical therapy. These remissions are of uncertain duration and frequency. However, I have not encountered a single instance of *chronic* distal ileitis in which remission has lasted for a number of years.

There has been considerable controversy in recent years as to what constitutes the most desirable operation in the treatment of distal ileitis. One school is of the opinion that all such patients should be treated by ileocolic resection in one or two stages with complete excision of the diseased ileum and the enlarged mesenteric lymph nodes, on the theory that the disease is primarily one of the mesenteric lymph system with secondary changes in the bowel. On the basis of an experience with the largest individual series of cases ever reported and on the basis of a most careful and thorough follow-up study, we are of the opinion that resection is rarely necessary and that the lowest mortality and best follow-up results are obtained by the simpler operation of ileotransverse colostomy with exclusion of (complete division) the ileum well proximal to the upper limits of the diseased segment. We believe that the disease starts in the ileum, that the lymph node involvement is secondary, that, following the simple short circuiting operation, complete recession of the node involvement takes place as has been demonstrated repeatedly at secondary laparotomy. The removal of the lymph nodes is no guarantee against recurrence because proximal recurrence has occurred even after the widest type of resection. We are of the opinion that ileocolostomy with exclusion is the operation of choice because, in close to 100 patients so treated, there has been no mortality, because, in all but three instances, abdominal wall fecal fistulas have closed spontaneously, because in every instance of fistula between ileum and bladder, spontaneous healing has taken place, because large right lower quadrant inflammatory masses have gradually been absorbed and, what is very important, because our follow-up studies have shown the smallest percentage of recurrences. It is possible to perform an operation with a zero mortality and produce follow-up results which are infinitely superior to those following the more radical and hazardous procedures. These methods involve extensive dissections of fistulous tracts and the opening up of large areas of retroperitoneal cellular planes with an operative mortality of 13 per cent and a recurrence

rate almost double that following the simpler operation.

About two years ago Crohn and I reported the results of a careful study of 164 cases of ileitis. We were particularly interested in the comparative operative mortality and the follow-up studies. Sixty-five patients were treated by ileocolostomy with exclusion with a zero mortality. Fifty-five patients were subjected to a one-stage ileocolic resection with a 16.3 per cent mortality. Twenty-five patients had a two-stage ileocolic resection with a mortality of 12 per cent. In nineteen instances of combined ileocolitis, a more complicated problem, the operative mortality was 10.5 per cent. The follow-up studies indicated that the ileocolostomy group showed a recurrence rate of 10.5 per cent, the one-stage ileocolic resection, a recurrence rate of 15.4 per cent and the two-stage resection group, a rate of 28.6 per cent. It seems to me that this analysis is conclusive proof of the superiority of the simpler operation of ileocolostomy with exclusion.

There are certain practical points in the operative management of ileitis that deserve emphasis:

It is an excellent plan to pass a Miller-Abbott tube preoperatively because it serves to decompress the bowel, thereby making for a smooth convalescence and aids in identification of various loops of bowel should extensive adhesions be encountered.

We recommend that the patient be placed in Trendelenburg position in order to aid in visualization of the various bowel segments.

Some years ago, I suggested that the abdomen be opened by a left rectus incision on the theory, that should a secondary resection become necessary at a later date, it could be carried out by a right sided approach in a field free of adhesions. The pathological lesion can be clearly visualized if the Trendelenburg position is used.

Under no circumstances should the surgeon attempt to separate the diseased bowel from viscera, such as bladder, cecum, sigmoid or small bowel, to which it has become attached, because such a maneuver might result in the opening of sealed-over fistulous tracts. The whole purpose of the operation would thereby be defeated.

The entire small intestine must be carefully inspected for "skip" areas. The finding of diffuse disease in ileum and jejunum calls for no further operative manipulation inasmuch as jejunoileitis is non-surgical. It is important to divide the ileum well proximal to the most proximal "skip" lesion.

The operation of ileocolostomy in continuity should never be performed. The ileum *must* be divided always in order to effect a complete by-passing of the diseased ileum. In spite of the fact that we have repeatedly emphasized this feature as the most important part of the operation, we have seen an appreciable number of patients in recent years where division was not practiced and the surgeon formed the opinion that, because the symptoms were not relieved, the operation was a poor one. As a matter of fact, if the ileum is not transected, the symptoms are aggravated because a closed cesspool is created between the anastomosis and the strictured ileum with the production of additional toxic symptoms caused by absorption of noxious products in the bowel. In four patients so treated that I explored secondarily simple division of the ileum distal to the anastomosis resulted in an amazingly dramatic change. At the risk of appearing repetitious, I would repeatedly stress the importance of division of the ileum.

For the average surgeon, a side-to-side ileocolostomy is probably the safest type of anastomosis.

The various pathological, clinical and therapeutic features of ileitis have been stressed in this report because it is felt that a clear understanding of the whole problem is needed if optimum results are to be obtained. Particular emphasis has been placed on the need for thorough exploration of the entire small bowel and the use of an operative procedure which will give the lowest operative mortality and the largest percentage of desirable therapeutic results.

MESENTERIC LYMPHADENITIS

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IT is generally conceded that the mortality of appendicitis is the mortality of delay, often brought about by uncertainty as to diagnosis. I believe that another possible factor is the fact that some medical men are of the opinion that many patients with appendicitis will recover in two to three days without surgery and, therefore, feel justified in delay.

This erroneous idea is sometimes based on the fact that they regard every attack of acute right lower quadrant pain as being appendicitis and fail to realize that other conditions may produce similar symptoms. Thus when they see a patient with symptoms that suggest the possibility of an appendicitis and at operation see only a harmless appearing organ, they may think that the operation was unjustifiable and that these symptoms can be taken lightly more often than not.

While this idea is not very prevalent, I have seen evidence of the fact that it does exist and I believe it, too, is a factor in the mortality of appendicitis. If a broader understanding of the other conditions which may produce symptoms similar to those of appendicitis is developed, the consideration of these conditions will tend toward greater accuracy in diagnosis and should be of some aid in lowering the present mortality figure.

Thirty-five per cent of appendices removed under the diagnosis of appendicitis are said to be normal. This does not include those removed incidental to some other abdominal operation, such as pelvic disorders or gallbladder disease. A 35 per cent of error as quoted by Arkush and Kosky is enough to make a number of medical men feel justified in delays which may prove disastrous in some of the 65 per cent which are not normal. We must remember that the mortality of acute appendicitis is still too high and that increasing knowledge and awareness of this condition by both doctors and public has not lowered it as much as we had hoped. Under these circumstances it is perfectly justifiable to remove an appendix whenever warning signals have been given, that made this diagnosis a probability.

There are some circumstances which make the conscientious medical man, or even the surgeon, who has had a recent appendiceal death hesitate to advise or to perform an appendectomy. One of these is a proper hesitancy to do abdominal surgery in the presence of appendiceal peritonitis. While all are not agreed upon this point, there are many high grade clinicians of wide experience, who practice conservative treatment at this time. Enough in fact to furnish adequate backing for anyone who prefers to follow expectant treatment in the presence of peritonitis.

At the other extreme, especially in children, is the condition noted when a child during the terminal phase of an upper respiratory infection develops abdominal pain and tenderness. The doctor who fears the dangerous complications of an appendicitis will have an appendectomy done, despite the sometimes greater danger of postoperative pulmonary complications and yet after taking this chance will find only a normal appendix. Two or three experiences of this sort will make him uncertain of his diagnostic acumen, and the next patient with somewhat similar symptoms, but this time with an inflamed appendix as the cause, may be allowed to go on to perforation.

Again, while most surgeons no longer accept the term of "chronic appendicitis," many children are seen who have periodic attacks of abdominal pain, associated with tenderness in the right lower quadrant. These subside quickly but often are found to recur after a mild cold, sore throat or slight bowel upset. If operation is decided

upon to relieve the recurring symptoms, an appendix will be removed which is normal grossly and microscopically, yet in a surprising number of cases the recurring symptoms disappear.

Of the several conditions which may be confused with appendicitis, the most common is that condition which has been termed "mesenteric lymphadenitis." While this condition has been referred to by some European writers, attention was called to it in this country by Dr. Leonard Freeman, of Denver, in an article presented to the Western Surgical Association in 1923. Since then, interest in it has grown, and a number of articles have been published, but it is still considered all too seldom in the diagnosing of acute abdominal conditions.

It first came to my attention in 1920 when Dr. C. U. Collins, of Peoria, Illinois, demonstrated it in a clinic he was holding and commented that when a person had symptoms of appendicitis but when at operation a normal appendix was found, the surgeon should carefully investigate the abdominal cavity until the real pathological condition was located; that in children enlarged mesenteric lymph nodes would often be found and that these were sufficient to account for the symptoms. He also stated the comforting fact that while the cause of the trouble was not certain, all the patients recovered from the operation and most of them from their symptoms.

The symptoms of mesenteric lymphadenitis have been recorded repeatedly for many years and several theories have been developed as to the cause. The consensus of opinion seems to be that the enlarged lymph nodes are due to absorption of toxins of bacteria. These bacteria have either caused the primary intestinal inflammatory reaction such as diarrhea, or to swallowed infected secretions in the course of an upper respiratory infection. The pain is presumably intestinal griping due to irritation from the same source.

The symptoms of mesenteric lymphadenitis are quite variable but in general are as follows:

Pain is the most constant symptom. It is usually colicky in nature and comes on with paroxysms which increases in intensity. In the beginning it is mild and not as severe as the average appendicitis. In a few cases we have noted symptoms of real severity and a small number were so severe as to resemble a kidney colic or an intestinal obstruction in severity. The pain is due to intestinal spasm, and when severe may be accompanied by audible peristalsis. This is not true in the average case but only in the exceptional one of unusual severity.

Nausea and vomiting were more common in the older patients, nausea in 60 per cent and vomiting in 25 per cent. Temperatures varied from 99°F. to 103°F., but the average, even after two to three days, was from 99.6° to 100°F. The white blood count varied in proportion to the fever, averaging around 8,000 to 12,000, but occasionally being much higher. The differential count frequently shows an excess of lymphocytes but this is so variable that it can not be regarded as being diagnostic.

Rigidity is found occasionally but is usually not present. Incidentally, it may be present at one examination of the patient and absent later.

The chief method of differentiating between mesenteric lymphadenitis and acute appendicitis, in our experience, is in eliciting abdominal tenderness. In the average case of acute appendicitis, the tenderness is localized to the right lower quadrant and is usually at McBurney's point. Rebound tenderness is quite constant, and the further away from McBurney's point one presses the less the tenderness becomes.

In acute mesenteric lymphadenitis, however, the tenderness is quite generalized. There may be a tender area over McBurney's point, but there is often another corresponding tender area on the left side. One side is often as tender as the other and a moderate amount of tenderness is found in the upper abdomen also. Rebound tend-

erness is less likely to be noted and the general impression is one of suspected appendicitis but with symptoms being too mild to let the examiner feel certain that the appendix is really at fault.

Occasionally, a patient with a fever of 102°F. is found, but when contrasted with one of acute appendicitis with this much fever a marked difference is found in the lack of prostration. The patient with a fever of 101° or 102°F., suffering from acute appendicitis looks and feels sick. The one with mesenteric lymphadenitis and this fever is not so hard hit.

The pathologic changes in the abdomen at operation vary according to the number, size and location of the lymph nodes involved. Sometimes they are enlarged throughout the length of the mesentery while in some they are limited to the ileocecal region. As a rule, individual nodes are seen scattered irregularly throughout the mesentery but sometimes they are so numerous as to appear confluent. They vary in size from that of a pea to that of a fifty cent piece although very few ever attain this size. We have never found suppuration or fluctuation. In patients with very severe symptoms some free fluid has been found in the peritoneum. At times the tissues about the nodes are injected and some nodes are adherent but they usually move freely between the layers of the mesentery. At times, particularly when a diarrhea has been present, even several days before the attack of abdominal pain, the terminal ileum has been quite injected. In these patients the lymph nodes were the most extensively involved of all.

In the course of the past twenty years this group has paid particular attention to this condition. We believe that it is a clinical entity and that given a reasonable amount of clinical experience it can be diagnosed in a high percentage of cases. In some seasons, especially in the spring months when respiratory infections are common, we have seen it run as high as 35 per cent in the patients in whom appendicitis was the original diagnosis.

Other than in providing academic interest in recognizing abdominal conditions, it has some very practical points of application. Whenever we see a child with an attack of acute abdominal pain, with tenderness most marked in the right lower quadrant with some fever and leucocytosis, we first and foremost consider the possibility of acute appendicitis. If the fever is slight and there is a relative lymphocytosis, and especially if the abdominal tenderness is somewhat generalized, we also give serious consideration to mesenteric lymphadenitis.

Our routine procedure is to try to arrive at a definite differentiation whenever possible, but we normally advise an immediate appendectomy. We have seen too many instances in which our preoperative diagnosis was mesenteric lymphadenitis but upon opening the abdomen a distended, acutely inflamed appendix was found. Whenever we open the abdomen with the diagnosis of mesenteric lymphadenitis and find a normal appendix, we are usually rewarded by the demonstration of numerous enlarged and often injected mesenteric lymph nodes. One consolation in this event is that many of these patients have given a history of repeated attacks of abdominal pain and we believe that we can be quite certain that not only will an initial recovery take place, but that the chronic abdominal symptoms or recurrent attacks of pain will no longer occur.

The exceptions to this statement are the children with large tonsils, cervical and inguinal adenopathy and a history of recurrent attacks of sore throat. In a few of these, but in only a few, the symptoms may recur until a tonsillectomy has been done and the gateway closed to further infection.

I believe that the most practical application of the diagnosis of mesenteric lymphadenitis exists in instances of upper respiratory infection with referred abdominal pain. In analyses of deaths from appendicitis it is not uncommon to find quoted instances in which children with

known respiratory infections have developed abdominal pain. An appendectomy has been done in spite of this existing pathological condition because of the fear of an appendicitis. Operation has revealed a normal appendix and autopsy has shown the pneumonia which was the real cause of death and which might not have developed had the abdomen been treated conservatively.

We in this group believe that when a child with upper respiratory infection develops abdominal pain it is well to consider the frequency with which mesenteric lymphadenitis complicates this condition. If this is done, the patient will not be so likely to be subjected to the additional risk of a laparotomy; for mesenteric lymphadenitis can be treated conservatively with perfect safety and in two to three days the patient will have recovered from the attack.

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SYMPTOMATIC ABDOMINAL EPILEPSY*

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IT is not a too infrequent experience for the surgeon or clinician to be confronted with a diagnostic dilemma and the decision whether to operate or not, when abdominal pain is the presenting symptom. In the majority of cases the symptom of abdominal pain is surrounded by other signs and symptoms with a recognizable sequence of events and an overall pattern which permit of the anatomic, etiologic and pathologic diagnosis. When these are partly wanting special laboratory or diagnostic procedures may be utilized to definitely establish the presence of intra-abdominal organic disease in its many forms. On the other hand many surgeons have felt the warm flush of embarrassment when faced with the realization of their diagnostic fallibility or impotence in certain cases, after removing an innocuous appendix or retreating empty-handed after an exploratory laparotomy. This situation usually arises in those cases in which abdominal pain has been a chronic symptom and which has eluded correct diagnosis after employing the numerous generally accepted diagnostic methods and which has not yielded to varied attempts at treatment. In this percentile small group which, however, is numerically large enough to justify and indeed require careful analysis before resorting to surgery or other ineffectual measures, there is to be considered the disorder known as symptomatic abdominal epilepsy.[1]

The true clinician will not make this diagnosis by exclusion but will require some criteria which are at least sufficiently clear to draw this condition into the pale of a recognizable entity. However, thus far, the consideration of symptomatic abdominal epilepsy, as a cause of abdominal pain has been a last resort measure, the usual story being one of prolonged, assiduous, extensive, futile studies of the gastrointestinal tract, other abdominal viscera, thoracic viscera, genitourinary tract, vertebrae, and spinal cord and its roots, with the final chapter often ending with a fruitless abdominal exploration or the patient being stigmatized with the appellation of psychoneurotic.

Definition. Symptomatic abdominal epilepsy is a disorder characterized by bouts of paroxysmal abdominal pain, the latter being due to hypermotility of the bowel, provoked by abnormal discharges of certain neurones in the vicinity of biochemically or structurally altered cerebral tissue, presumably situated in the premotor, and postmotor cerebral cortex and/or the diencephalon. The attack of abdominal pain at times may be single and prolonged only when the symptom occurs as the result of a vascular ictus, or a neoplasm involving the frontal and parietal lobe and/or diencephalon.

Experimental and Clinical Data. The most definitive recent work relating abnormal gastrointestinal motility with cortical influence has been by Fulton and his co-workers,[2] Spiegel, Weston and Oppenheimer,[3] Penfield and Gage,[4] and Penfield and Erickson.[5] In a brilliant series of animal experiments Fulton et al.[2] demonstrated that stimulation of area six (Brodmann) produced disturbances in gastrointestinal motility; ablation of the frontal lobes followed by hypermotility of the stomach and pyloric spasm enduring several days; and acute intussusception with fatal obstruction following bilateral ablation of the frontal lobes or its parts. These investigators were of the opinion that areas three

* From the services of the Jewish and Doctors Hospitals, Philadelphia, Pa. The histopathologic sections are from the John L. Eckel Laboratory of Neuropathology and were used through the courtesy of Dr. N. W. Winkelman.

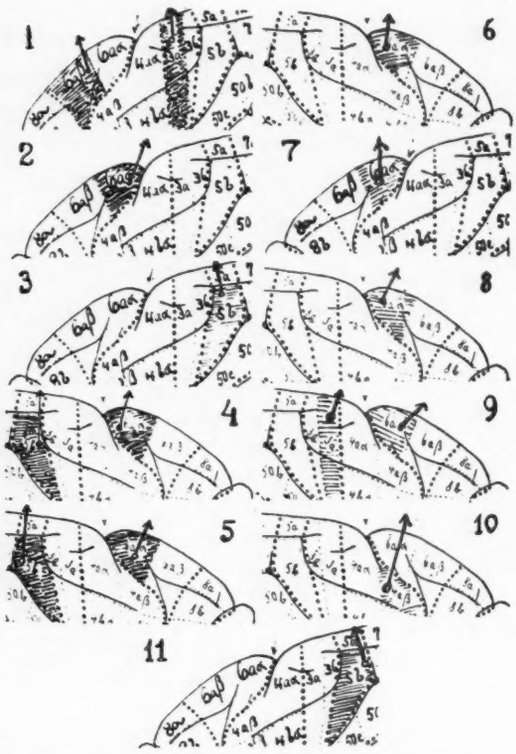


FIG. 1. Location of foci from which the gastrointestinal system could be influenced. The foci are indicated by the cruciate sulcus by (cortical areas in the dog according to Kelmpin) (SPIEGEL, E. A., WESTON, K. and OPPENHEIMER, M. J. Courtesy of authors and *J. Neuropath. & Exper. Neurol.*)

and five of Brodmann were also concerned with gastrointestinal activity. Penfield and Gage[4] stimulated electrically area five-a of the exposed brain of a patient, resulting in pain in the lower right quadrant of the abdomen which then was followed by a convulsive seizure. It was shown quite clearly in experiments on dogs by Spiegel, Weston and Oppenheimer[2] that the whole gastrointestinal tract could be influenced by stimulation of areas six, five and three. (Fig. 1.) They observed that stimulation of these areas may produce effects in all parts of the gastrointestinal tract synergically, increase of activity in one part may be associated with inhibition in other parts of the gastrointestinal system, or occasionally only a limited part of the bowel may be influenced.

The exact manner in which the diencephalon exerts its influence upon gastrointestinal motility is not entirely understood. However, Watts and Fulton[6] have produced experimental diencephalic lesions resulting in disturbed gastrointestinal activity. Cushing[7] called attention to the probable rôle played by the diencephaon in disturbances of gastrointestinal motility and the production of peptic ulcer following brain trauma when there was involvement of the diencephalon.

Clinically, it has been known for some time that abdominal distress or even pain may constitute part of the preconvulsive aura of epilepsy and Fulton[8] has stated that this pain usually coincides with vigorous abnormal gastrointestinal movements. Abdominal pain likewise has been reported as a symptom of gross cerebral disease. Wechsler[9] has seen patients who were treated for some time and even operated upon for abdominal syndromes in whom abdominal pain was the symptom of a cerebral lesion. This pain can closely resemble the pain of gallbladder disease, gastric or duodenal ulcer, appendicitis or renal colic. Winkelman[10] cited the case of a forty-five year old man who experienced intense abdominal pain as the initial symptom of a cerebral hemorrhage involving the frontoparietal areas. Abdominal pain in children frequently is an extremely difficult symptom to evaluate and recent studies by Klingman, Langford, Greeley and Hoefer,[11] and also by Lambert[12] have shown that formerly unexplained attacks of paroxysmal abdominal pain were a manifestation of the convulsive state. These workers were able to observe cerebral dysrhythmias in the electroencephalographic studies of their patients of the petit-mal and psychomotor equivalent varieties. Some of the children had associated symptoms of the convulsive state accompanying the paroxysmal abdominal pain.

In his very succinct and inclusive way, Hughlings Jackson said, "Epilepsy is the name for occasional, sudden, excessive, rapid and local discharge of gray matter."[13] The expression of subjective or objective manifestations in the patient depends upon the locus of discharge. The commonly seen

motor Jacksonian convulsive seizure is often the symptomatic expression of a focal discharge in the motor area resulting from a discrete lesion, be it tumor, traumatic scar or other morphologic structural change. Should the lesion which initiates paroxysmal cerebral dysrhythmias be situated elsewhere in the brain, the symptoms would reflect the function of that part. It is, therefore, not an exercise of the imagination to conceive of focal lesions involving any one or all of areas six, five and three of the cerebral cortex or of the diencephalon which could produce, under appropriate conditions, abnormal discharges which would lead to disturbed gastrointestinal activity and consequent abdominal pain. These lesions could very well be of varying magnitude in size and of different histopathologic appearance.

In order to demonstrate the relationship existing between abdominal pain as a symptomatic expression of cerebral disease, the representative cases cited here will take into account those lesions which are gross and demonstrable, those in which a history of pre-existing disease or trauma would conceivably lead to a cortical lesion, and finally those in which there may be some question as to a specific causal factor producing a focal lesion with subsequent abnormal cortical discharges.

Case 1. (Brain tumor—single focal lesion). H. P., was a sixty-three year old male whose chief complaints consisted of periodic attacks of severe epigastric pain and headache. He was first seen by me on April 21, 1946. For six to eight weeks prior to this he had complained of recurrent attacks of intense abdominal pain which led to complete *x*-ray study of the gastrointestinal tract by his attending physician. These proved negative. Loss of weight directed attention to possible malignancy, but none could be unearthed. About five weeks prior to examination he developed headaches situated over the frontal and suboccipital regions. Shortly thereafter a pronounced mental change occurred which was interpreted as a depression and the diagnosis of involutional depression was made by several physicians. He was to be transferred to a mental institution to receive electroshock treatment when I was requested to see him.

On the day of examination he showed the following; mental hebetude, spontaneous, brief outbursts of unprovoked crying, mild left hemiparesis, suggestive hesitancy of the left leg in walking, wavering of the outstretched left hand, increase of the deep tendon reflexes of the left arm and leg as compared with the right side, characteristic grasp reflex in the left hand, suggestive Babinski phenomenon on the left, mild weakness of the left corner of the mouth and bilateral choked discs. When questioned as to the most distressing symptom he pointed to the "pit of his stomach." Spinal puncture revealed a pressure of 320 mm. C.S.F. *X*-ray examination of the skull was negative. A diagnosis of tumor of the right frontal lobe lying rostral to the motor gyrus impinging on area six was made. In view of the rapid progress of symptoms it was believed that the lesion probably was a glioma.

On April 30, 1946, a craniotomy was performed by Dr. Robert A. Groff at the Graduate Hospital of the University of Pennsylvania. Directly beneath area six at a depth of about 2 to 2½ cm. below the pial surface a discrete spherical tumor mass was encountered and removed. (Fig. 2.) The overlying and surrounding brain tissue was soft and appeared swollen. The tumor mass was firm, showed a smooth pseudocapsule and measured 2.1 cm. in diameter. Histologic study revealed the cytologic appearance of a metastatic malignant growth with some characteristics resembling spongioblastoma multiforme. The surrounding white matter and overlying cortex showed a pronounced glial proliferation. Postoperatively the symptoms of headache and abdominal pain disappeared. The pronounced forced grasping quickly diminished within three days after operation.

This case illustrates the occurrence of paroxysmal abdominal pain which had been interpreted as a form of intrinsic disease of the gastrointestinal tract. The early phase of this patient's symptomatology was exclusively gastrointestinal. The subsequent headache apparently had been dismissed in its significance and later even the gastrointestinal symptoms, after the *x*-ray findings had been negative, were likewise dismissed as a somatic complaint

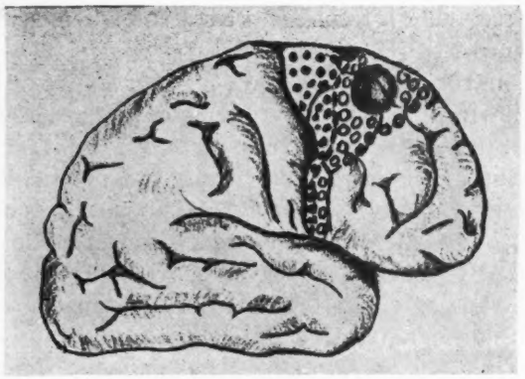


FIG. 2. Sketch of right side of brain indicating the position of the tumor found in Case 1, solid circles motor area 4; open circles pre-motor area 6.

part of the picture of an involutional depression. The mental status of the patient should have been considered, as it was later, as a symptom of organic brain involvement. Had this been done and the headache properly evaluated, the symptom of abdominal pain would have completed the pattern establishing the diagnosis of a brain tumor involving the right frontal lobe. The abdominal pain, therefore, was the symptomatic expression of abnormal discharges arising in the premotor area secondary to the "irritation" or stimulation by a localized space-taking lesion subjacent to area six.

CASE II.[14] (Tuberous sclerosis—disseminated lesions in the brain.) A. D., female, aged three years, was admitted to the Jewish Hospital with a diagnosis of rickets. At the age of five months she had recurrent attacks of abdominal pain which caused her to "double-up" and cry out. These seizures were of relatively short duration and occurred both during the day and at night. The attacks of abdominal pain were followed by excessive urination and sleep. Studies of the gastrointestinal tract revealed nothing abnormal. The attacks of abdominal pain endured until the patient was eleven months of age. A tentative diagnosis of intestinal tetany was made and the patient treated on this basis. Subsequently the child failed to develop in a normal manner and was unable to talk, had no control of bowel or bladder function and showed abnormal behavior with periodic, abrupt attacks of crying, screaming and thrashing about. These attacks

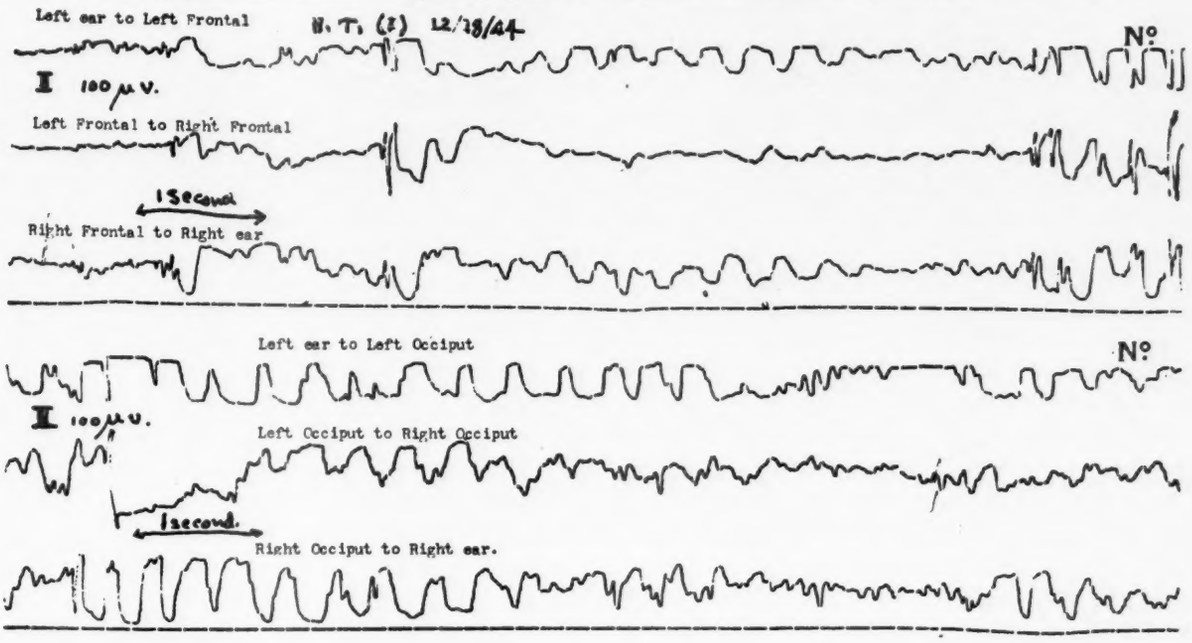
FIG. 3. Section of brain showing disseminated lesions of tuberous sclerosis. (Weil stain × 2.)

were unpredictable and occurred at irregular intervals during the day. When examined neurologically the positive findings were pronounced mental deficiency, partial atrophy of both optic discs, defective hearing, nevus multiplex of Bourneville (sebaceum adenoma of Pringle) over the nose and maxillary region. Complete laboratory studies consisting of *x*-ray examinations and chemical studies of the blood and spinal fluid proved negative. The paroxysmal nature of the abdominal pain from the age of five months to seventeen months was interpreted as an epileptoid manifestation since these were replaced by recurrent bouts of behavior disorder having the pattern of the convulsive state.[15] The triad of mental deficiency, nevus multiplex of Bourneville and epileptic phenomena allowed the diagnosis of tuberous sclerosis.

The nodules of tuberous sclerosis are irregularly scattered throughout the brain and not only can they involve the motor area producing the usual epileptic motor seizures observed in tuberous sclerosis but they undoubtedly also involve premotor area six and the postcentral areas five and three. In the case cited here the lesions provoked abdominal pain as part of the total picture of an epileptic display. Figure 3 shows the irregular distribution of the lesions of tuberous sclerosis and how they implicate the cerebral cortex producing abnormal discharges from "irritated" cortical ganglion cells.

CASE III.[16] (Cerebral trauma.) N. T., male, age eleven years, gave the history of having had three attacks of unconsciousness during the four years prior to examination on December

Fig. 4. (Case III). Electroencephalographic tracing prior to treatment. (Courtesy *J. A. M. A.*)



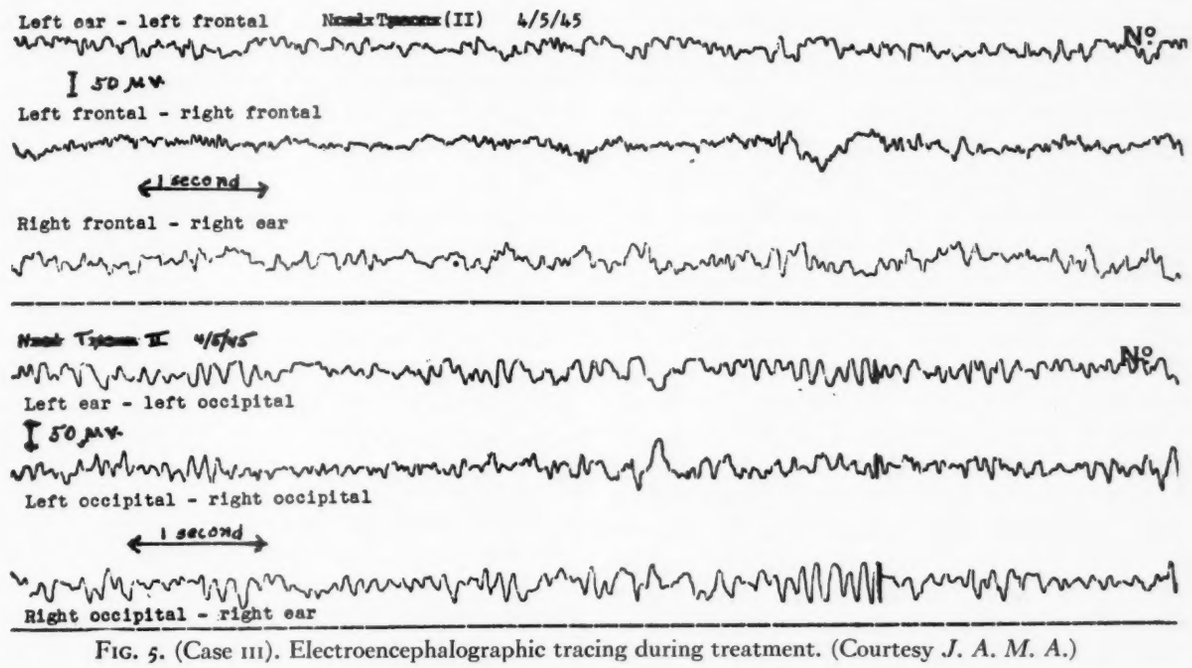


Fig. 5. (Case III). Electroencephalographic tracing during treatment. (Courtesy *J. A. M. A.*)

11, 1944. There were numerous interval seizures of abdominal pain, nausea, occasional vomiting and diarrhea. The abdominal pain came on intermittently and consisted of "waves" of abdominal pain occurring about twice weekly. The individual attacks of abdominal pain would last from two to three minutes and appeared at intervals of from two to ten minutes, the entire "wave" lasting approximately one hour. These "waves" of pain appeared either in the early morning or at night and were not associated with unconsciousness or diarrhea. He had one severe attack of abdominal pain in July, 1944, which endured ten minutes and which was followed by vomiting and diarrhea. During the year preceding examination he had frequent nightmares and talked in his sleep. This patient had sustained a severe head injury five years before. While sledding down an incline he struck his head and was unconscious for twenty-four hours. The outstanding symptoms which fol-

FIG. 6. Cortical damage due to craniocerebral trauma. (Toluidin blue stain × 100.)

lowed the head injury were recurrent episodes of abdominal pain interspersed with occasional convulsive seizures and unconsciousness. It was believed that the entire constellation was a form of epilepsy resulting from injury to the cerebral cortex. Electroencephalographic studies as shown in the illustration (Fig. 4) revealed slow waves which could be seen in all leads. These slow waves seemed to predominate and were more persistent in the frontal areas. It was concluded by Dr. Donald Scott, the encephalographist, that "the record most closely approximated the pattern seen in idiopathic epilepsy."

This patient was subsequently placed on an anti-convulsant therapeutic regimen consisting of dietary control, supervision of activities, and medication in the form of diphenylhydantoin sodium (dilantin sodium) gr. 1½ twice daily and calcium-bromido-galactogluconate (Calcibronat—Sandoz) 2 Gm. dissolved in water three times daily. Immediately after being placed on this form of therapy the attacks of abdominal pain ceased and to the present writing, one year and five months after treatment was started, this patient has had no abdominal pain and no grand mal seizures. Subsequent electroencephalographic tests (Fig. 5) with the patient on medication showed the usual level of normal activity with groups of slow waves both with and without spikes. The latter occurred most prominently in the frontal and occipital regions. This record showed 30 per cent less abnormal activity than the previous study.

This patient had had three attacks of incomplete convulsive seizures during a five-year period following severe head injury. The predominant feature during that interval had been recurrent attacks of abdominal pain coming on at frequent intervals. These had been misinterpreted completely and eluded treatment until anti-convulsant therapy was instituted, since which time there has been a complete cessation of symptoms.

This case illustrates the relationship between cerebral injury producing cortical damage and the resultant abnormal discharges from the adjacent cortical neurones, as graphically demonstrated by the electroencephalographic studies, resulting in paroxysmal abdominal pain (due to involvement of the frontal and/or parietal lobes) or convulsive seizures (due to implication of the motor cortex). The photomicrograph (Fig. 6) shows the degree of cortical damage which may result from cerebral trauma, and the ganglion cells in the immediate periphery of such lesions may be the origin of abnormal discharges capable of being picked up by the electroencephalograph.

CASE IV.[17] (Angioneurotic edema of the brain with focal cortical damage.) P. P., male, aged thirty-seven, was first seen by me in June, 1941. The chief complaint was the single

symptom of periodic attacks of severe cramp-like pain involving the entire abdomen. The pain often started in the lower right quadrant and then involved the whole abdomen. Individual attacks of pain endured up to two hours or more and were intense and unremitting. During the attack there was a "rumbling" sensation in the abdomen. When the attack of pain subsided, he was exhausted and felt "achey all over." The bouts of pain began at the age of nine months and occurred at irregular intervals, appearing every seven or eight days for months preceding my original examination, and during the week preceding the examination four seizures had occurred. The first attack came on shortly after the patient had received diphtheria antitoxin, which had made him quite ill. When eight years of age an appendectomy was performed during one of the seizures and a normal appendix was removed. At the age of sixteen he injured himself and anti-tetanus serum was given. He became extremely ill and experienced one of his usual attacks of intense abdominal pain. He also had urticaria and a high degree of fever for two days thereafter. There was a history of development of hives after eating certain foods. This patient had been studied thoroughly in many institutions over a period of practically the entire range of his illness. The types of treatment were many, but at no time did he obtain lasting relief. The longest attack free interval was fourteen months. An objective neurological examination failed to reveal any evidence of overt disturbance of the nervous system. Complete studies with respect to cytology, blood and spinal fluid chemistry, *x*-ray studies of the gastrointestinal tract, gallbladder and urinary tract, all proved negative. The blood pressure was 102 systolic, 68 diastolic, pulse rate 76, weight 139½ pounds. During the study of this patient it occurred to the examiner that the symptom of abdominal pain, in view of its irregular occurrence, paroxysmal nature, duration of the seizure, and the post-seizure statement by the patient that he was "exhausted and felt achey all over," a frequently observed phenomenon in the postconvulsive phase of epilepsy, represented a form of epileptic disturbance.

In order to ascertain the correctness of this point of view, two steps were pursued; the first consisted of having electro-encephalographic studies performed, and the other to place the patient upon an anti-convulsant regimen. These studies revealed the presence of abnormal low voltage waves seen mainly in the right and left frontal regions. They were also present in the parietal and occipital lobes. Hyperventilation increased this abnormal activity. (Fig. 7.) Anticonvulsant medication was begun during the middle of June, 1941. This consisted of phenytoin sodium (dilantin sodium) 1½ gr. (0.1 Gm.) twice daily and a bromide mixture, 1 fluid dr. (4 cc.) four times daily. For a period of over seven and one-half months he remained attack free, following which he was requested to stop medication in order to determine whether the seizures would return. Within a period of less than a month, he developed a severe attack. Medication was resumed and he remained attack-free for seven and one-half months, at which time the dilantin sodium was replaced by lactose in the white and red ringed capsule and the bromides replaced by sodium chloride. This was done without the patient's knowledge in order to eliminate the element of suggestion. Fifteen days later he developed the characteristic seizure. He was informed about the experiment after his extreme alarm at the reappearance of the attack. This patient has remained attack free from July, 1942, to the present writing, April, 1946, with only two exceptions which occurred during the latter part of 1945 when the patient had grippe and stopped taking the anti-convulsant medication, and during the early part of 1946 when he had again lapsed in the use of dilantin sodium and the bromide mixture. During the period of three and one-half years between July, 1942, and December, 1945, he was entirely free of seizures and took the dilantin sodium faithfully and the bromide mixture somewhat irregularly. Electro-encephalographic studies performed during the time he was on active treatment (Fig. 8) showed that the cerebral potentials in both hemispheres were within normal limits. There was an occasional abnormal sharp wave. Large, slow, abnormal waves could be brought out in both frontal lobes after hyperventilation.

This patient had an exquisitely defined symptom of abdominal pain without any preceding phenomena and following an attack only the experience of exhaustion and achiness. The question as to whether this might be abdominal migraine had been eliminated diagnostically, first because this diagnosis had been made in

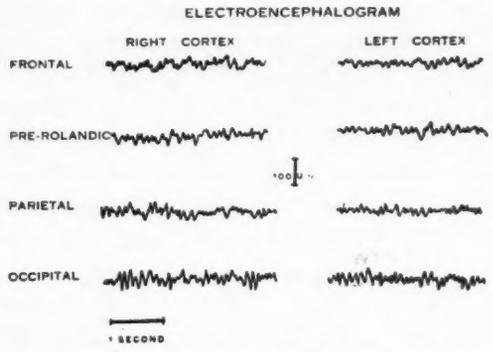


Fig. 7. (Case iv). Electroencephalographic tracing prior to treatment, showing low voltage three per second abnormal waves in the right and left frontal cortex. They are present to a lesser extent in the parietal and occipital areas. (Courtesy *J. A. M. A.*)

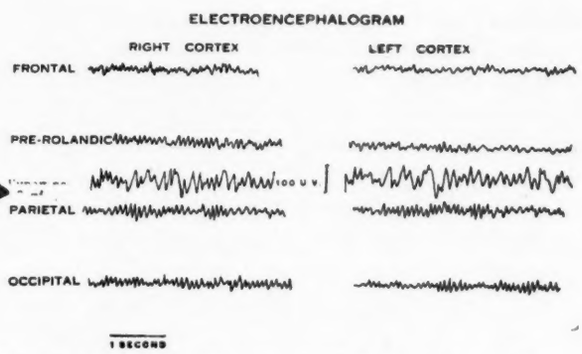


Fig. 8. (Case iv). Electroencephalographic tracing during treatment with anticonvulsants. The cerebral potentials throughout both hemispheres are within normal limits as to amplitude and rhythm. After hyperventilation, denoted by arrow, large slow abnormal waves are detected in both frontal lobes. (Courtesy *J. A. M. A.*)

one of the hospitals where he had been examined and the use of ergotamine tartrate failed to prevent attacks or abort one in progress. Furthermore, the usual history of abdominal migraine was absent. That is, other migranious phenomena failed to appear such as hemicrania preceding or following the attack of abdominal pain, visual disturbance, nausea, vomiting or diarrhea. The pattern of the occurrence of abdominal pain coming on suddenly, at irregular intervals, and presenting a stereotyped form, suggested the epileptic nature of the disorder. It was believed that the disturbance probably arose in those cortical areas of the brain subserving gastrointestinal function and that the original use of serum, when the patient was nine months of age, probably had produced angioneurotic edema of the brain with some alteration of structure in the areas stated. That such a point of view is justifiable is based upon the experimental and clinical observations of Dechaume and Croizat[18] and Bassoe,[19] and the objective electroencephalographic findings of abnormal activity in the frontal and parietal lobes. Dechaume and Croizat[18] experimentally produced congestive, hemorrhagic and degenerative changes in the brain following serum shock in animals. The case reported by Bassoe[19] of a woman who developed aphasia, hemiplegia, hemianopsia and convulsive seizures following the injection of serum, showed upon exploratory craniotomy an area of dark, hyperemic softened brain tissue, which was regarded as the effect of a focal angioneurotic edema of the brain.

This case illustrates the development of the symptom of paroxysmal abdominal pain in an individual in whose brain changes had occurred following the use of an antiserum. Specifically, angioneurotic edema is postulated to have occurred with subsequent damage to the cortical neurones of the cerebral cortex, mainly in the frontal lobes and to a lesser degree in the parietal lobe. These areas of altered tissue have provoked a cerebral dysrhythmia as seen in the electro-encephalographic studies, and when the appropriate factor or factors were present, such as the reaction set up by certain foods to which he had been shown to be allergic, the process of summation in the irritated ganglion cells was invoked eventuating in the explosive discharge producing hypermotility of the bowel and abdominal pain.

Case v.[20] (Isolated symptom of paroxysmal abdominal pain without demonstrable etiology.) P. P., a married female, aged forty-four, complained of bouts of abdominal pain, beginning twelve years prior to the date of examination on May 4, 1944. Two years after the onset of her repeated abdominal pain, she had her appendix removed. The latter showed no morbid process. The attacks began with a peculiar sensation of "gnawing in the abdomen" which was then followed by generalized pain. The latter was accompanied by a "rumbling noise in the intestine." The abdominal pain at times was associated with a "funny, dazed feeling" and at such times she

feared that she was going to "pass out." The attacks of abdominal pain appeared for the most part at night. She always maintained consciousness throughout these seizures. During the twelve-year span there were three occasions, all of which occurred while in bed, in which the abdominal pain was associated with a "shaking" of her limbs and jaw, which she was unable to control voluntarily. The latter episodes lasted from ten to fifteen minutes and would awaken her from sleep. There were no premonitory symptoms other than the "gnawing" sensation cited above. The seizures occurred about twice weekly for the four-month period preceding examination. Her attack free intervals usually lasted no longer than one week. She had been married nineteen years and the past medical history revealed no data which would throw any light on the possible etiologic cause. From the time of her appendectomy, she had consulted numerous physicians and was ultimately labelled a psychoneurotic. A review of her personality development and the psychiatric examination showed no disturbance in her emotional or mental spheres. There was no evidence of organic disturbance of the nervous system following a detailed neurological examination. *X*-ray studies of the gastrointestinal tract and laboratory studies showed normal findings during the attack free intervals. Physical examination of the chest and abdomen likewise was negative. Electroencephalographic studies revealed no abnormal cerebral discharges.

Despite the fact that this patient presented no previous history of cerebral injury or disease predisposing to changes in the brain or meninges, it was believed, because of the several facts, such as the periodicity of the attacks, their sudden explosive onset, the occasional aura-like introduction by a "gnawing" sensation in the abdomen, the "funny, dazed feeling" and the pseudoconvulsive movements which had occurred in a few of the seizures, that when pieced together, these fitted into the pattern of an epileptic display. This patient was put on an anti-convulsant regimen, the medication consisting of hydantoin sodium 1½ gr. (0.1 Gm.) twice daily and phenobarbital gr. ¼ (16 mg.) three times daily. Within one week after the institution of treatment on April 24, 1944, the attacks of abdominal pain disappeared and she has remained attack free since that time.

The only elements in this case which appear to warrant and justify the diagnosis of abdominal epilepsy are (1) the epileptic pattern of the aura-like manifestations, the manner of occurrence of the abdominal pain and the accompanying epileptoid symptoms; (2) the immediate and continued response to anti-convulsants.

COMMENTS

The five cases which have been summarized in brief fashion represent a variety of causes which may produce the symptom of abdominal pain as an expression of altered changes of gastrointestinal motility effected by the remote disturbances occurring in the cerebral cortex, mainly in areas six, five and three. They also represent the illustrative cases of a larger series in which the most frequent cause appears to be craniocerebral injury.[21]

Case 1 demonstrates perhaps most clearly a specific locale as to the origin of abnormal discharges provoking the symptom of abdominal pain. In this case the abdominal pain was first thought to be of intrinsic gastrointestinal origin. The surrounding historical data, such as headache, mental change, and the neurological findings of increased intracranial pressure, hemiparesis, grasp reflex, altered tendon reflex activity, etc., led to the consideration of a cerebral neoplasm. The operative finding of a discrete tumor lying directly beneath area six in the cortex, would seem to be ample indication of a causal relationship between "irritation" of the neurones in this area and the symptom of abdominal pain. The explanation for the other frontal lobe signs and symptoms, such as mild motor weakness, grasp reflex, occasional hebetude and tendon reflex phenomena, may be based on the glial reaction and swelling of the tissue surrounding the discrete tumor mass. The topographical situation of the tumor in this case clinically bears out the experimental findings of Fulton and his co-workers[2] and of Spiegel et al.[3] Unfortunately, time and lack of facilities did not permit of electro-encephalographic studies

being performed in this case. Had they been done, however, it is not at all unlikely that focal abnormal discharges would have been picked up at the periphery of the lesion in area six.[22] It is only a step, therefore, beyond this to interpret positive, abnormal electroencephalographic findings from the frontal lobes and/or the parietal area in cases in which abdominal epilepsy exists as evidence of an intrinsic disorder of, or morphologically altered, cerebral cortex in areas six, five, or three. The alterations of structure in these regions may be due to one of numerous conditions, most of which have already been well outlined by Cobb.[23]

For purposes of illustrating the mode of study in this type of disorder I shall cite merely a few conditions which may be predisposing factors in the syndrome of abdominal epilepsy. The existence of a gross lesion such as tumor, abscess, intracerebral hemorrhage, thrombosis of a cerebral vessel, are both clinically and pathologically demonstrable as involving either the frontal or parietal lobes. When the symptom of abdominal pain occurs under such circumstances, it can be attributed to stimulation of cortical areas six, five or three. At times the abdominal pain is the direct result of massive stimulation of the neurones in these areas as in an ictus due to hemorrhage or thrombosis. However, in brain tumor, as in the case reported here and in cases in which the pain was paroxysmal, there must, therefore, be an explanation other than continuous "irritation" which provokes the "seizures of pain," just as there must be some explanation for the periodic fits in the classical form of grand-mal epilepsy. An effort at explanation of this will be offered later. Brain tumor is a notable cause of epileptic fits as has been shown by an extensive literature on the subject.[24] The cases of cranio-cerebral injury previously reported by me[1] in which the symptom of paroxysmal abdominal pain was an outstanding feature, I believe to be a demonstration of cortical scars occurring in areas six, five, or three. The illustration of the cerebral cortex (Fig. 6) of an individual who sustained a cranio-cerebral injury, shows the damage to the functioning cortex and the definite morphologic changes which occur. The functioning neurones at the periphery or in the vicinity of such a lesion may and do give off abnormal discharges, which under appropriate conditions may be of such magnitude as to constitute an adequate stimulus to an effector structure. The scars consequent to brain injury can be seen directly at operative exploration.[5] They also frequently can be demonstrated by electro-encephalographic studies after a definite history of brain injury is in evidence.

It is well established both clinically and neurohistopathologically that infections of the brain and the meninges, especially the latter, may produce fixed tissue changes which may ultimately manifest themselves in disturbed function of the affected brain in the form of convulsive seizures. Here then, is another cause which, if the lesion or lesions occur in areas six, five or three, may produce the symptom of abdominal pain either singly if the lesion is exquisitely placed, or in association with other manifestations of the epileptic state.

Disseminated lesions of the brain offer by mathematical chance a greater likelihood of one or more of the lesions being placed in areas six, five or three, producing the symptom of abdominal pain. Case 11 illustrates this. Other conditions such as multiple sclerosis in which the brain has become involved, Schilder's disease, toxoplasmosis, multiple cerebral arteriosclerotic scars and the like, may also involve areas six, five or three, producing the symptom of abdominal pain, usually in conjunction with other symptoms reflecting the function of the involved brain. Figure 9 illustrates the nature and extent of a cortical arteriosclerotic scar which conceivably could be a single scar involving one of the cortical areas under discussion. Under such circumstances a patient could have the isolated symptom of paroxysmal abdominal pain, and clinically one can imagine the

many conditions which probably would be considered first in the way of intrinsic abdominal disease before relating the symptom of abdominal pain to a possible cerebral cause. Other conditions provocative of structural cerebral change may be toxins of various types. Specifically metallic poisonings, the main offender being lead, may produce changes either of a minimal degree or more extensive disseminated changes in the brain. The form of lead encephalopathy encountered in infancy or childhood producing mild symptoms, may ensue in recovery, because of the relatively minimal damage to the brain. Figure 10 shows an area of the cerebral cortex of a case of lead encephalopathy in which there is a small focal alteration with destruction of neurones, and changes in the ganglion cells in the immediate vicinity of the focal devastation. It will be seen that the endothelial nuclei of the capillaries are swollen and where they oppose each other cause a slowing up or stoppage of blood flow with resultant dropping out of cells, and ischemic cell changes of other neurones in the area supplied by the vessels.[25] It is from such lesions which may or may not ultimately cause electroencephalographic abnormalities and which may not be surmised unless an extremely careful history is obtained, that the symptom of abdominal pain may occur either singly or in association with other larval or aberrant displays of epilepsy.[15] This together with other noxious agents, such as the cerebral reactions to sera, vaccines, high fever and the secondary encephalopathies incident to measles, scarlet fever and other infections[26] constitute a group of causes which may occur at any time after the birth of the individual, and which if the lesion or lesions be situated as already mentioned, the symptom of abdominal pain can and does occur. Intrauterine existence does not protect the fetus from contracting some type of cerebral affection which may ultimately produce a form of epileptic disorder. It has been shown that the cerebral lesions of toxoplasmosis begin in utero[27] and clinically

Fig. 9. Arteriosclerotic scar producing "puckering" of the cerebral cortex. (Klarfeld stain.) × 65.)

this condition is characterized, among other things, by convulsive seizures. Should lesions in this condition affect only cortical areas six, five and three, one can see the causal relationship with abdominal pain. Other conditions of ante-natal origin[28] are likewise known to result in epileptic seizures.

A less easily demonstrable, but equally important consideration of what may be a factor in unexplained paroxysmal abdominal pain occurring both in children and in adolescents may be the minimal neurohistological changes which are produced in the brain of the fetus secondary to febrile or toxic conditions occurring in the mother during the period of gestation. Figure 11 shows a focal area, one of many, which occurs in the brain of an individual suffering from toxemia of pregnancy. There are changes in the small vessels, particularly the capillaries, in which there is endothelial thickening, resulting in focal areas of cellular devastation and severe cell disease. Other areas may show ischemic cell

FIG. 10.

FIG. 11.

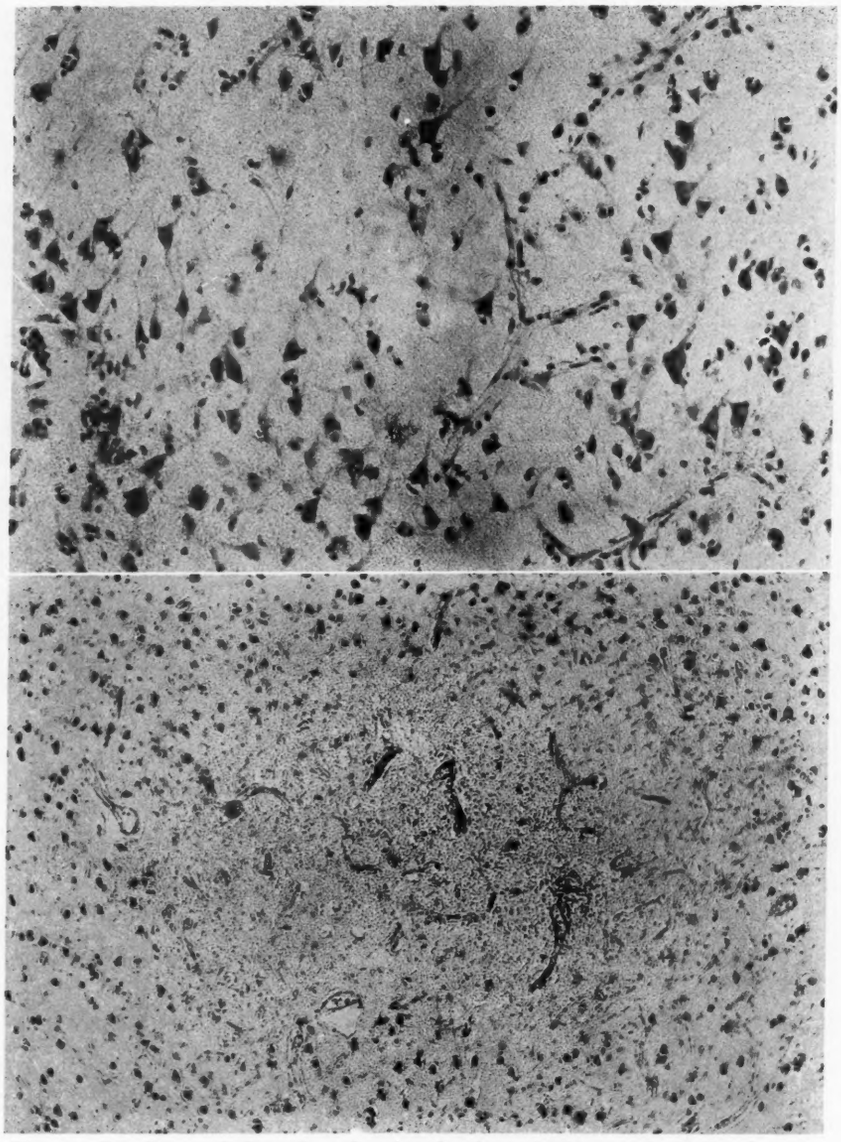


FIG. 10. Lead encephalopathy, showing focal cortical area of cellular devastation, intimal changes in the capillaries and cell disease of the adjacent ganglion cells. (Toluidine blue stain × 210.)

FIG. 11. Toxemia of pregnancy, showing focal cortical damage, with an acellular area, capillary proliferation and gliosis. (Toluidine blue stain × 210.)

disease secondary to the disturbance of circulation in the small vessels. The biochemical disturbances which constitute the condition known as toxemia of pregnancy are operative not only on the cerebral tissues of the mother, but certainly must affect the more vulnerable brain of the fetus also. In those individuals in whom toxemia of pregnancy subsides under proper treatment and where the delivered infant survives, it is not taxing credulity to visualize at least a few such areas, as shown in the illustration, in the brain of the surviving offspring.

Again, braving the criticism of repetition, a lesion of this sort in areas six, five or three presumably could eventuate in disturbance of gastrointestinal motility, giving the clinical picture of abdominal epilepsy, or perhaps in other forms of gastrointestinal syndromes, which to the present have escaped acceptable explanation. Further study along the lines of electroencephalographic findings in various gastrointestinal

disorders, characterized by changes in motility, may yield fruitful information with respect to cerebral disorders. This tenuous hypothesis may be borne out somewhat by the case previously reported by me,[1] in which the forty-three year old patient, over a period of many years, had had abdominal symptoms consisting of paroxysmal abdominal pain, "rumbling" of the bowel and diarrhea. These gastrointestinal manifestations were at times associated with vasomotor phenomena and occasional unconsciousness and muscular twitchings. He had been considered at various times as having enterocolitis, duodenitis, spastic colon and the like. Treatment with an anti-convulsant regimen provided relief for the first time since the age of twelve when the attacks were initiated.

MECHANISM OF ABDOMINAL EPILEPSY

The various factors concerned in the initiation and discharge of an adequate stimulus from cerebral neurones to effector structures resulting in paroxysmal abdominal pain are in no way different from those operative in the generally accepted forms of symptomatic epilepsy. A discussion of the literature and theoretical aspects of the mechanism of the epileptic discharge would lead too far afield and finally would reveal only the open question as to the ultimate nature of the "trigger" factor or factors.

Electroencephalographic abnormalities as a graphic reflection of underlying cortical disturbances have been reported in conditions too numerous to mention here and range from the gross space-taking lesion to the finer lesions of microscopic size.[29] In an electroencephalographic study of four cases of encephalitis and meningoencephalitis, Ross[30] showed non-specific electroencephalographic abnormalities in the acute phase of the disease. He also indicated that the abnormal discharges may persist and be the harbinger of epilepsy.

In 1924, Harris[31] reported five cases of spontaneous hypoglycemia wherein the symptoms of excessive hunger, weakness, nervousness and anxiety were predominant, and in several patients abdominal distress was an added outstanding feature. Bulatao and Carlson,[32] in experimentally insulin-induced hypoglycemia in man and animals, observed an increase in gastric tone and in the height and frequency of contractions gradually developing into tetany. The abdominal pain thus induced could be either generalized or localized depending on the extent of bowel stimulated. Hoefer et al.[33] studied a group of twenty-seven patients having verified islet cell adenoma of the pancreas, with respect to the neuropsychiatric manifestations of "convulsions, coma, and related states of impaired consciousness." Among twenty-two of these patients having "autonomic, visceral symptoms," four had abdominal pain. Eleven of these had electroencephalographic studies and eight records taken during the fasting state showed abnormal discharges, among which were synchronous bursts diffusely scattered of the spike-and-wave variety, and others of the slow activity.

Extensive studies by Gellhorn[34] have indicated that an "anoxidative state" in the brain is induced by hypoglycemia, metrazol convulsions, injections of sodium cyanide and inhalation of high concentrations of carbon dioxide. Gibbs, et al.[35] stated, "nitrogen breathing, standing with a lowered blood pressure and over-ventilation, all of which produced large, slow waves in normal subjects, also tend to precipitate seizures in epileptic persons." Putnam and Hoefer[36] have shown, by means of the electroencephalograph, abnormal cerebral discharges in conditions associated with lowered metabolism of the cerebral neurones with special reference to anoxia, and disturbances of carbohydrate metabolism. They made the significant observation "that depression of metabolic function may precipitate an increase in cerebral activity under a variety of con-

ditions and that impaired consciousness and increased neuronal activity are compatible." Thus any of the numerous physiologic and organic disorders, which disturb carbohydrate metabolism, or which induce cerebral anoxia, may lead to abnormal discharges from cerebral neurones.

It will be seen from the foregoing briefly submitted data that two groups of conditions can provide a milieu conducive to setting off abnormal electrical discharges from cerebral tissue, capable of being perceived electro-encephalographically. The first is concerned with definite structural alterations of brain tissue of varying types and causes which may, under "appropriate" conditions, "fire off" and provide an epileptic display. The second group deals with biophysiochemical disturbances which, in the final analysis, produce a deficient oxygen up-take and impoverished carbohydrate supply to the functioning neurone.

It is believed, therefore, that in those fixed morbid tissue changes of the brain the ganglion cells in the vicinity of the lesion may give off a continuous subliminal discharge; and when bodily disorders, whatever their origin, provoke the second factor of disturbed oxygen and carbohydrate metabolism of the brain, which likewise may induce a subliminal discharge, the latter thereby constitutes the "appropriate" condition which by "summation" provides the "adequate" stimulus to an effector organ. In the present case the effector organ is the terminal motor-swelling in the smooth muscle of the bowel. The pain is due to the disordered motility of the gastrointestinal tract and is transmitted by the afferent projection pathways.

Method of Study. 1. *Anamnesis:* This should be painstaking, detailed and exhaustive, and should include all historical data pertaining to the progenitors, physical state of the mother during gestation, evidence of birth trauma, allergic phenomena during and subsequent to infancy, infectious diseases of childhood and all other infections, craniocerebral injury, endocrine and metabolic disturbances, and all other organic conditions which required medical or surgical treatment. The symptom of abdominal pain should be considered with respect to age of onset, frequency and rapidity of appearance, time of day (pre- or post-prandial), duration, association of pre- or post-seizure sensory phenomena, accompanying symptoms usually associated with the epileptic state, or epileptic phenomena antedating the presenting episodes of abdominal pain, and symptoms referable to increased intracranial pressure or active cerebral disease or dysfunction. I cannot emphasize too strongly the value of an adequate history, which in most instances should often suffice to indicate the proper direction toward correct diagnosis.

2. Each patient should be examined intensively, by the indicated available methods, to rule out the existence of (1) intrinsic disease of the abdominal, pelvic and thoracic viscera, (2) metabolic or endocrine disorders, (3) morbid involvement of the spinal cord or peripheral nerves, and (4) psychosomatic projection mechanisms.

3. Thorough neurological examination and complete laboratory investigation, should, when indicated, include cytologic and chemical studies, roentgenologic studies of the skull,—both direct and contrast (pneumo-encephalography and thorotrast cerebral angiography) and electro-encephalographic studies, should be employed to disclose the presence of some structural or physiologic disorder of the brain residing in cortical areas six, five or three and/or the diencephalon.

4. Finally, resort to the empirical method of the therapeutic test may be the only or corroborative evidence of the existence of abdominal epilepsy. Should the symptom of paroxysmal abdominal pain disappear or be radically altered by an anti-convulsant regimen, recur upon cessation of treatment and again yield to active therapy, the requirements of an ac-

ceptable therapeutic test will have been met.

Treatment. When the collective data reveal the presence of an intracranial space-taking lesion, surgical intervention, in operable cases, is indicated. Focal traumatic scars likewise should be excised, when the symptom of paroxysmal abdominal pain and any attendant phenomena cannot be controlled by an anti-convulsant regimen. Those conditions which may disturb carbohydrate metabolism and/or oxygen supply of the cerebral neurones thus providing the biochemical "trigger factor" in the production of abdominal pain, such as hyperinsulinism, hyperthyroidism, congestive heart failure, Adams-Stokes disease, intoxications, allergies, blood dyscrasias and the like, should receive treatment specifically directed. If cerebral syphilis is uncovered during the study for the cause of abdominal pain the obvious treatment is anti-luetic. Foci of infection should be eradicated and attention should be directed to factors which might precipitate reflex afferent stimuli.

The fixed morphologic cerebral changes and the biochemically altered cortical cells, already alluded to and not amenable to surgical treatment, which act as the sources of abnormal electrical discharges producing paroxysmal abdominal pain should be treated by the following anti-convulsant regimen: (1) The use singly or preferably in combination of (a) diphenylhydantoin sodium (dilantin sodium), (b) phenobarbital, (c) bromides—alkaline or calcium bromido-galactogluconate (calcibronat). These drugs are to be used in adequate dosage depending upon age, weight and habitus of the patient; (2) high vitamin, high protein, moderate fat, moderate carbohydrate diet; (3)controlled liquid intake, adequate to meet the daily needs of a balanced water metabolism. This means neither dehydration nor water intoxication; (4) supervised social, physical and emotional activities within reasonable limits, designed to establish an even tenor of living.

SUMMARY AND CONCLUSION

Attention has been directed to a form of abdominal pain which represents the symptomatic projection of distant cerebral disease or dysfunction.

Experimental and clinical data are presented indicating the relationship of abnormal gastrointestinal motility and abdominal pain, with lesions affecting cortical areas six, five and three and/or the diencephalon.

Allusion is made to some of the various morbid organic changes, and other disorders which may result in less easily demonstrable neuropathologic conditions, which may provoke abnormal electrical discharges in those areas subserving gastrointestinal function, thus providing the stimulus for altered motility of the bowel ending in the symptom of abdominal pain.

Representative cases are cited to illustrate the location of definable gross cerebral lesions capable of visual observation, and others, due to a variety of causes, demonstrable by electro-encephalography, which are believed to be the origin for the symptom of paroxysmal abdominal pain.

An effort is made to describe and define, with sufficient inclusiveness, a syndrome which provides the essentials for definitive diagnosis without resorting to a diagnosis by exclusion; and a tentative explanation of the mechanism of symptomatic abdominal epilepsy is submitted.

The method of study and treatment are outlined.

The symptom of paroxysmal abdominal pain, when properly fitted into the pattern of historical events and symptoms, in the light of the foregoing presented data, may be considered not only as a form of symptomatic abdominal epilepsy but also may serve as a guide-post in the topographical localization of cerebral lesions.

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Disruption or eventration of abdominal wounds usually occurs between the seventh and eleventh days after operation, after the use of vertical incisions closed with catgut. Wound infection, drainage, cough or other strain favors the disruption. After a secondary closure, a secondary eventration may occur.

From "Principles and Practice of Surgery" by W. Wayne Babcock (Lea & Febiger).

PREOPERATIVE AND POSTOPERATIVE CARE OF ACUTE SURGICAL CONDITIONS OF THE ABDOMEN*

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THE surgeon long ago established his art upon the basic fundamentals of asepsis, hemostasis, gentle handling of tissues, astute diagnostic ability and personal integrity. The past fifteen years have brought many improvements in surgical technic but the reduction of surgical morbidity and mortality has been greatest since these fundamentals were combined with skilled preoperative and postoperative care. The more acute the surgical condition, the more applicable becomes this statement. Thus is affirmed the pertinent fact that surgical technic must not be divorced from surgical theraupetics. This is the thought Sir William Osler sought to convey when he stated, "A surgeon should be a physician who operates." Careful preoperative and postoperative care can virtually fulfill Lord Moynihan's desire that, "Surgery has been made safe for the patient and now the patient must be made safe for surgery." There are very few surgical emergencies that do not permit fundamental preoperative care and all surgical patients merit enlightened postoperative care. This applies to every operative procedure as not one is of "minor" surgical status. Surgery of any nature can be a dangerous method of therapy.[1]

PREOPERATIVE TREATMENT

When a patient suffering from an acute surgical condition of the abdomen presents himself, the surgeon does more than evaluate the primary disorder. It is necessary to weigh the severity of pain, amount of anxiety, possibility of shock, presence of dehydration, existence of hemorrhage, probability of infection, the nutritional state and concurrent disease. To answer and thus effectively deal with these problems an inventory must be taken. This includes a history, physical examination and a laboratory investigation.

Inventory. This period, short though it may be, allows for careful surgical planning. The surgeon seeks to secure the patient's confidence and cooperation.[2] Failure to prepare a patient psychologically may complicate skilled surgery or minimize its benefits. A methodical history not only aids in revealing the proper diagnosis but provides an orderly, useful source of material for later analysis. It may ward off complications by the patient revealing such conditions as a drug idiosyncrasy, alcoholism or a state of hemophilia.[3] In this manner the physician not only wins the patient's regard by his interest and thoroughness but is rewarded by facts that contribute to a purposeful diagnostic pattern.

A physical examination should be a well planned, conscientious search for every vestige of information pointing to the patient's specific complaint and his general state of being. More diagnostic errors are committed from lack of observation, or slipshod interpretation than from not knowing the disease *per se*.[4] Too frequently an examination includes only the region of apparent pathological disorder. Do not formulate a diagnosis on impression; gather facts, all the facts. During an examination it is well to have the patient alone, well covered, as comfortable as possible, in good light and with the bladder and rectum empty. The examiner should gently proceed with an orderly, complete scrutiny, keeping his eyes and ears alert. Examine the suspected area last. Record at once in writing the findings and impressions. Such

* From The Department of Surgery of The Jackson Clinic, Madison, Wisconsin.

a commitment stands later as irrefutable evidence of one's diagnostic skill or of its lack.

The laboratory offers valuable assistance in the preoperative examination. Certain tests will be utilized to evaluate the specific surgical complaint, others to reveal the patient's general surgical status. For example, an elevation of the blood's nonprotein nitrogen may necessitate a careful genitourinary tract investigation, or call for dietary or medical management; it may influence the choice of anesthesia and the extent of purposed surgery. A hemoglobin value below 60 per cent may be ominous.[5] The presence of an elevated blood pressure is cause for vigilance; a very low blood pressure is cause for concern. A normal electrocardiogram is a vote of confidence but not insurance against cardiac complications. It is in this manner the surgeon evaluates the many aids the laboratory can offer. When properly selected a laboratory investigation can reasonably predict a patient's surgical tolerance.

Immediate Preoperative Period. This period covering from a few minutes to several hours must be expertly utilized as every minute counts. The surgeon need be explicit in his instructions to his surgical assistants and the nursing staff. Standing orders which are generally applicable to patients with acute surgical conditions of the abdomen include:

Record your observations of the patient's pertinent actions and comments.

Call the laboratory at once for desired tests and assist in obtaining specimens.

Give nothing by mouth and aspirate (do not wash) the stomach if food has been recently taken.

Pass a Levin catheter through the nostril and institute gastric suction.

Oral hygiene is instigated and false dentures removed from the mouth.

Select and indicate the most acceptable vein for intravenous fluid .

If intravenous fluids are to be started, (1) select a vein not likely to be disturbed, (2) introduce a No. 19 gauge needle, (3) secure it firmly in place, (4) do not lower the fluid flask or pinch its tubing to check the needles position.

Catheterize female patients and leave a clamped catheter in place.

Catheterize male patients if a residual is suspected. Leave catheter in place if the surgical procedure is apt to prove prolonged.

One and one-half hours before surgery exclude visitors from patient's room.

Cover the eyes with cotton and place cotton fluffs in the ears; hold all in place by a towel fitted to the head.

Administer preoperative medication and request quiet of roommates and hospital personnel.

Note and record patient's general response every thirty minutes thereafter.

Chart, *x*-rays and special equipment are made ready to accompany the patient.

Deliver the patient to the surgical room at the appointed time.

Preoperative Medication. The choice of preoperative medication is largely determined by the constitutional state of the patient being operated, the procedure to be executed and the anesthetic to be used. Properly administered preoperative medication should relieve the patient's anxiety, reduce or eliminate the excitement stage of anesthesia, minimize the amount of anesthetic required, lessen the tendency to shock and render recovery possible with less effort and fewer complications.[6] There is no one drug or combination of drugs that is ideal. The amount used must be individualized and under no circumstance should amounts be routinized.[7] This applies especially to the oft used "quarter and hundred and fiftieth."[8] Divided doses of preoperative medication are to be preferred to hazarding overdosage. Basal anesthesia should never be allowed to so depress or confuse a patient that he cannot identify himself just before receiving an anesthetic.[7] All drugs available for preoperative sedation may produce idiosyncracies. Morphine is probably the most frequent offender although probably the most useful drug. Given hypodermically it requires at least

one hour to exert its maximum effect although this action can be obtained in ten minutes if given intravenously.[8] Barbiturates are especially valuable due to their soporific effect and ability to modify the toxic qualities of a local anesthetic.[7,9] They are more useful from the standpoint of comfort than they are safe, however.[8] Scopolamine may occasionally produce periods of excitement. Hyoscine may predispose to more postoperative complications than related drugs.[9,10] Paraldehyde is a safe and effective drug which may be given orally or rectally.[11] The use of atropine sulfate, although time honored, does not merit the tendency to its routine use. In acute abdominal surgery the Jackson Clinic finds frequent use for sodium phenobarbital one and ½ gr. orally or 3 gr. rectally one and one-half hours before surgery. One hour before surgery pantopon ⅓ gr. and often scopolamine $\frac{1}{150}$ to $\frac{1}{300}$ gr. are given hypodermically.

Skin Preparation. The skin site of a purposed surgical incision should receive careful attention. Gentle cleansing with mild solutions is sufficiently effective to render undesirable harsh scrubbing and strong solutions.[12] The presence of hair in an operative field may not be as surgically offensive as numerous skin nicks made in its unskilled removal. It is well to prepare a large skin area.[4] Open wounds, the umbilicus and sinuses may be sealed with collodion. Exercising aseptic technic and utilizing white soap, sterile water and cotton balls gently scrub the skin for two minutes. Next shave the area cleanly with a sharp blade, wash away all loose hair and continue gentle scrubbing for eight more minutes. Use plenty of white soap. Rinse the skin with sterile water. If a tinted germicidal dye is desired zephiran (1:000) or merthiolate[13] are permissible. Skin prepared as just described is as well prepared as by any method. The skin should be prepared just before surgery. In the event that more than an hour elapses after the above preparation and before surgery the skin merits another five minute scrub just before draping using the above technic. It is well to remember that the skin is not rendered "sterile" by any ritual of preparation, but the method just reiterated has physiological merit and proven virtue.

SPECIAL PREOPERATIVE SURGICAL PROBLEMS

Poor Risk Patients. Many patients who must submit to surgery due to an acute abdominal complaint are poor risks. This condition may be due to the primary disorder but it is often the result of an associated state.[14,15] The "poor risk" cannot always be anticipated but the very young and the aged are generally not good risks.[16] Children require greater surgical care as they tolerate pain poorly, are emotionally unstable, cooperate reluctantly, are unpredictable in accepting nourishment, fall easy victims to infection and have water, acid-base and nitrogen equilibriums that are readily disturbed.[16] These tendencies demand careful preventive measures. The aged surgical patient is becoming more prevalent. When handled carefully they tolerate surgery well but are more prone to circulatory and renal failure, pulmonary complications, embolic phenomena and slow tissue repair.[17]

Diabetic Patients. Joslin states that over 50 per cent of all diabetic patients receive surgical care some time in their life.[18] Statistical studies further show that the number of diabetics has doubled in the United States in the past decade.[18] In emergency surgical cases particularly accompanied by infection the operative risk and mortality are greater in the diabetic.[19] Every effort must be made to control the diabetes as certain symptoms of diabetic acidosis (i.e., nausea, vomiting, abdominal pain and leucocytosis) may simulate or mask an acute surgical condition of the abdomen. Not infrequently an acute abdominal lesion may precipitate a diabetic acidosis.[18] When this occurs, combat dehydration and acidosis with large amounts of fluids, glucose and insulin.[20] Keep in mind that the most prominent symptoms of protamine hypoglycemia are nausea, vomiting,

headache and dizziness,[21] thus rendering differentiation from acidosis at times difficult. Protect the patient with a high carbohydrate intake covered with sufficient insulin as judged by blood sugar determinations. The diabetic subjected to surgery especially when infection is present will demand substantial increases in insulin.[18,21,22] Spinal anesthesia is ideal for the diabetic patient. Once operated upon the diabetic becomes an excellent candidate for postoperative complications.

Cardiac Patients. Patients with suspected or recognized cardiac lesions cause the surgeon considerable concern. The blood pressure and the electrocardiogram frequently do not aid in evaluating the heart's work capacity. Very often a more valid and simple cardiac test is the heart's response to exercise or the patient's reiteration of his ability to exercise. It is necessary to keep in mind the often wide latitude between heart disease and heart failure. It is true that surgical patients with angina pectoris, syphilitic aortitic insufficiency, aortic stenosis and heart block may be subjects for sudden death.[23] Render the cardiac patient free of pain with an opiate, alleviate anxiety by a barbiturate and promptly fortify the heart muscle with digitalis when indicated. During surgery be doubly vigilant to avoid a fall in the blood pressure.[24] Following surgery instigate measures to prevent distention and vomiting, assist the patient to move freely, give parenteral fluids slowly and in well tolerated amounts and do not allow the patient to exert himself. The inhalation of oxygen spares the heart, reduces respiratory effort, hastens the recovery of the nerve centers and assists in preventing distention and pulmonary congestion.

Gastrointestinal Patients. Acute disturbances of the gastrointestinal tract early exert a profound biochemical disturbance in the patient. Special evaluation must be given to the presence or absence of obstruction, the electrolytic balance, store of protein and the presence of infection.[25] When acutely involved the gastrointestinal tract must be placed at rest by withholding oral nourishment, emptying the stomach through a Levin catheter and when indicated decompressing the small bowel by means of a Miller-Abbott tube. The value of the Abbott-Rawson tube for simultaneous gastric suction and intestinal alimentation is recognized. In event barium has been administered as a diagnostic medium see that it is removed before surgery. Preoperative catharsis has no place in these cases.[11] The value of an enema utilizing a mild solution has its indications. These patients, due to reduced oral intake, vomiting and diarrhea, suffer from water and electrolytic balance disturbances calling for careful but often copious amounts of parenteral fluids. Hypoproteinemia may produce obstructive edema of intestinal suture lines and should be corrected by whole blood and amino acids. The need for additional vitamin C and vitamin B complex is present and vitamin K is of value even though jaundice is not present. Too frequently these patients receive inadequate calories during the first seven, even fourteen days postoperatively. Do not calculate the nourishment intake in terms of cubic centimeters of fluid but rather in calories derived from Grams of carbohydrate, fat and protein.

POSTOPERATIVE TREATMENT

Immediate Postoperative Period. The period immediately following surgery and for four hours thereafter is an especially vital one for the patient.[14] This is all the more true if a general anesthetic has been given. The value of a surgical recovery ward devoted exclusively to postoperative care for the patient's first twelve to thirty-six hours is recognized. In its absence adequate standing orders in the hands of competent lieutenants are essential. Orders applicable to the immediate postoperative period of acute abdominal cases include:

Transferal from operating table: Do not suddenly alter a position that has been long maintained.[1] Support the spine in its physi-

ological position. (The anesthetist at this time will supply a listing of the patient's general state, blood pressure, pulse, respirations and medications recently given).

Transportation on operative cart: Do not leave the patient alone for a moment. Keep him well covered. See that the patient's chart accompanies him.

Transfer from cart to bed: Have the bed warm and flat. Lift, do not drag the patient into bed. Cover lightly (excessive covering contributes to restlessness and water loss by sweating).[26] Do not place pillows beneath the knees (they invite vascular complications). A moderate Trendelenburg posture is useful (it aids cerebral circulation, lightens the cardiac load and facilitates bronchial drainage).

Maintain a free airway: Have at hand a mechanical airway, tongue forceps, mouth gag, emesis basin and towel.

Inventory of the patient's condition: Note and record at least every thirty minutes for three to five times the patient's blood pressure, pulse, respirations, etc. Watch dressings for soilage, and properly connect drainage tubes.

Special equipment: Intravenous fluids, gastric suction and intranasal oxygen are used routinely; have the equipment ready.

Assist in recovery of motion: As a patient awakens from a general anesthetic he will often be confused and given to moving about. This is favorable to recovery and sedation should not be given in this "restless" period. Turn or assist the patient to do so. Move the limbs through their range of motion. Encourage deep breathing and gentle coughing.

Bladder care: Aid the patient to urinate. Do not delay catheterization especially when intravenous fluids have been given. When catheterization must be repeated request orders for special bladder care.

Oral hygiene: Moisten the lips, allow the mouth to be rinsed and the teeth to be brushed. Gum may be chewed; later glucose candy sucked upon.

Reassure the patient: Good nursing care allays apprehension, has a sedative effect and favors the return of physiological processes.

Postoperative Pain. Pain is often the first companion of a patient postoperatively. It is usually preceded by discomfort, restlessness, fatigue and insomnia. It is more readily controlled early in its onset than when anxiety and exhaustion become its comrades.[27] When not definitely contraindicated surgical patients deserve sufficient anodyne to provide comfort during the first two or three postoperative days. Morphine or one of its fractions (i.e., pantopon) is the best drug. Demerol is to be recommended for its anodyne and antispasmodic virtues. Such medications do not lend themselves to "p.r.n." ordering or as a compensatory medium for inadequate nursing care or rough surgical technic. Skilled nursing care and judgment in administering an anodyne contributes much to the prevention and to the control of pain. Too frequently a hypnotic drug (i.e., chloral, paraldehyde, bromides, barbiturates, etc.) is administered in hopes that it will relieve pain. These drugs are designed to produce sombulance not to relieve pain. The excellent pain alleviating qualities of acetylsalicylic acid are useful.

Fluids Postoperatively. It is not valid to assume that a patient can readily survive a postoperative period of nutritional withdrawal by calling upon stored constituents. It is true that a preoperative surplus of water, salt, carbohydrate, protein, fat and vitamins fortify in part a patient's postoperative needs, but only for a few hours.[28] Ideally all these substances are given orally; however, when this route is not available they are given rectally, subcutaneously, intravenously, intramuscularily, intraperitoneally and so forth. In certain instances one or a combination of these routes may be superior to others. Do not become wedded to any one method. Too frequently the safe, economical and, when properly used, easy administration of rectal nourishment is neglected. Intravenous alimentation is widely utilized and the sub-

sequent comments pertain to this method of fluid administration.

Water. Water constitutes 70 per cent of the body weight and stands second only to oxygen as a vital physiological substance.[29] The amount of water received by a surgical patient is as important as the dose of any other potent therapeutic agent.[30] Water participates in two major functions, namely, the elimination of waste (via the urine and the bowel) and the dissipation of heat (via sweat and the respiratory tract). The former has first claim upon the bodies water and the latter upon that which remains.[31] The water a surgical patient will require depends upon the amount vaporized, passed as urine, used to replace abnormal losses (i.e., vomiting, drainage, etc.) and that needed to overcome a previous state of dehydration.[32] The amount of water necessary to fulfill these needs is not easily ascertained. A number of laboratory tests (i.e., hematocrit determination, blood specific gravity, blood plasma's electrolytic pattern, etc.[33]) are of assistance but they are not specific. Certainly the surgeon should not wait for clinical symptoms of dehydration to signal the need for water. Nor should he rely upon an order to "force fluids" which is an unscientific request and if carried out literally could be dangerous.[32]

A gross but workable estimate of water balance can be secured by the careful measurement of the amount of fluid consumed and the amount of urine excreted. An output of 500 cc. may be adequate provided the urine is concentrated to a specific gravity of 1015 or above. Urine whose specific gravity is below 1015 may require 1500 cc. to complete its excretory task.[32] The average patient will require at least 2,000 cc. of water each twenty-four hours and certain patients may need twice this amount. The amount of water retained within the body depends upon the "holding" qualities of the electrolytic ions (i.e., sodium, potassium, calcium, chloride, etc.).[15,34] When these components are low, water promptly passes through the renal mechanism and is lost, when present in excessive amounts water is fixed in the tissues and edema may result.

Salt. The normal salt requirements varies from 5 to 10 Gm. daily which maintains a blood chloride level near 560 mg. per 100 cc. of blood and more than 1 Gm. in the urine. The major functions exerted by salt are: (1) maintenance of the acid-base balance, (2) retention of fluid in cellular tissue, (3) maintenance of tone in the intestinal musculature and (4) it is bactericidal.[5] The need for salt is markedly increased in the presence of pyrexia, vomiting, diarrhea, aspiration or loss of small bowel content, previous dehydration, etc.[32] Symptoms of salt deficiency (hypochloremia) develop when the blood level falls below 450 mg. per 100 cc. The symptoms may include mental depression, fatigue, nausea and stupor.[35]

So-called physiological saline has become a favorite solution for parenteral administration either alone or as a vehicle. The popularity of this solution is questionable when given in more than 2,000 cc. quantities. One thousand cc. of a 1 per cent solution of sodium chloride supplies the average need of 10 Gm. of salt. The merits of a hypotonic saline solution (i.e., 0.45 per cent sodium chloride) are evident when it is recognized that isotonic and hypertonic sodium chloride solutions may cause fluid withdrawal from the intracellular compartments.[36] Ringer's solution has special indications when potassium and calcium are needed.[31] Hartman's solution is one of choice in the presence of acidosis.[5]

Glucose. Glucose administered intravenously is a ready source of food; it helps prevent acidosis, protects the kidneys by sparing the bodies protein, assists the liver to maintain a glycogen balance, acts as a diuretic and is an essential metabolite in muscle contraction, especially of the heart.[5] A 5 per cent glucose solution in water is isotonic and provides about one hundred calories per each liter. The body has no difficulty utilizing glucose except when given too rapidly or when a state of diabetes is present. Winslow has shown that the daily

administration of 3,000 cc. of 5 per cent glucose in distilled water given at a rate of 300 to 500 cc. per hour is well tolerated and 98 per cent utilized. (It is well to remember that when 5 per cent glucose is added to a 1 per cent solution of sodium chloride a hypertonic solution results). When additional calories are desired a 10 per cent glucose solution may be utilized although it may temporarily cause tissue dehydration and resulting thirst.

Protein. The necessity of protein for the post-surgical patient has long been recognized but an adequate source and mode of administration is not easily available. The incidence of hypoproteinemia in post-surgical patients is high.[37,38,39] Those especially prone to develop this condition are patients with bleeding peptic ulcers, cancer of the gastrointestinal tract, bowel obstruction and intestinal fistula.[40] In the presence of hypoproteinemia fluids tend to leave the blood vessels and the administration of large amounts of sodium chloride solution only accentuates this process. Signs of hypoproteinemia may include lassitude, asthenia, slow wound healing, ascites, edema of suture lines and abdominal distention.[24,35,41] Blood plasma is a fair source of protein but whole blood is superior due in part to the value of hemoglobin as a protein source. The cost of either substance to combat hypoproteinemia is high as 10,000 cc. of whole blood may be required. To maintain a normal nitrogen balance following surgery as much as 70 Gm. of protein are required each twenty-four hours.[39] (1,125 cc. of blood plasma provides approximately 70 Gm. of protein). An amino acid solution prepared by the hydrolysis of casein is available in a 5 per cent concentration. (Amigen is an example). About two-thirds of the contained protein is available (approximately 33 Gm. per 1,000 cc.).[35] The solution is in general well tolerated, although relatively expensive and low in protein when used as its sole source. To such a solution may be added glucose, salt, vitamins and sulfanilamide.

Elman[38] states "That for surgical or other patients not able to take food by mouth, a simple plan may be drawn up in which all of the nutritional elements can be introduced intravenously in a volume of 3,000 cubic centimeters. This amount of water is probably necessary in most cases for maintaining water balance and providing a urinary output of about 1000 cubic centimeters or more per day. Of the three liters, one liter at least should contain five per cent protein digest and five per cent dextrose. Of the other two liters one will contain five per cent dextrose in water and the other isotonic solution of sodium chloride, which can be injected subcutaneously, thus relieving the patient of the long period required for the administration of three liters of fluid into the vein."

Vitamins. The added need for all the vitamins by surgical patients is well established. Virtually all may be given other than orally. Certain members are more essential than others, namely, vitamin K, C and the B complex. The need for vitamin K (menadione) in the newborn, the pregnant female, jaundiced persons and certain patients with atopy is recognized.[42] To operate a patient with the prothrombin below 60 per cent of normal is hazardous. The daily dosage of menadione is 2 to 4 mg. It may be given orally, intravenously or intramuscularly. When given orally supply 1 to 3 Gm. of bile salts daily. Cevitamic acid deficiencies occur in starvation, ulcers of the gastrointestinal tract, gastritis and other states. Besides symptoms related to scurvy its deficiency may prevent the proper deposition of collagen in a surgical wound.[14] Vitamin C should be given these patients in amounts ranging from 500 to 1000 mg. daily, taken orally or parenterally. The members of the vitamin B complex are especially indicated in deficiency states, alcoholism, central nervous system disturbances, diabetes, hyperthyroidism and following copious amounts of intravenous fluids. Their dosage under acute surgical circumstances must excede the therapeutic ones. In administering water

soluble vitamins fractionate the dosage or a wasteful renal spill will occur.[43]

Oral Nutrition. Following the opening of the abdomen a functional physiologic ileus develops. Its duration depends largely upon the extent and duration of trauma (either chemical or physical) that the abdominal tissues have sustained. Its presence is indicated by an absence of peristalsis and its disappearance by a return of the intestines' normal motility as determined by auscultation. This return may require as long as eighteen hours but this period can be shortened by gentle operative handling of tissues and the judicious administration of oral nourishment. Early feeding tends to decrease so-called gas pains, lessens the possibility of ileus and may assist in preventing a mesenteric thrombosis. The withholding of oral nourishment usually has little place in modern surgical care unless the taking of food jeopardizes the entrigity of the intestinal tube itself. In the event a long period of withheld oral nourishment is anticipated as after a gastrectomy, a tube (Abbott-Rawlson or plain catheter) may be introduced through the wound and into the intestine and used as an avenue of nourishment. The problem in every case, however, is an individual one not given to routine consideration. Should nausea develop, vomiting result or intestinal distention ensue it is well to withhold oral nourishment but in their absence oral nutrition may be given shortly after surgery. Liquids of preferance include hot water or tea followed by fat-free broth, gelatin, cold water and semi-solid food. By the third day a selected general diet is usually tolerated. Fluids prone to provoke distention include grape juice, orange juice, milk and certain flavored carbonated beverages. Frequently a patient has a consuming desire for a certain food which is often well tolerated when other substances may be rejected. Early oral nutrition often assists the bowel in producing a spontaneous stool circumventing catharsis and enemas.

Ambulation. Sufficient evidence is available not only to recommend early rising of surgical patients but to demand it. This practice was suggested by Emil Reis of Chicago early as 1889 and followed extensively abroad. It is now receiving its righteous recognition in this country as a major surgical contribution. The empirical use of eight or ten days of postoperative bed rest is in general traditional and unphysiological. Many surgical complications are the direct outgrowth of immobilization associated with bed rest. This is especially true when the abdomen and lower thoracic region is encompassed by a tight binder. Surgical incision plus the binder splint the abdominal muscles, limit the diaphragmatic excursion with resulting tendency to pulmonary complications, reduced cough reflex and lessened vital capacity.[44] Circulation in and about the wound is inhibited and tissue repair proceeds more slowly. Leithauser demonstrated that patients permitted to rise early following surgery regain their normal vital capacity in two to seven days while those kept in bed require seven to fourteen days. Certainly it is unphysiological to place tissues at rest if their circulation is thereby diminished and slowed. If circulation is slowed, healing is delayed, clotting is facilitated and embolism becomes likely. If disuse causes tissues to lose their full functional purpose, stimulate them and prevent this loss by assigning them their physiological task early in surgical recovery.[45] True, early rising presupposes certain surgical rules, namely, adequate pre- and postoperative care, careful hemostasis, accurate, gentle wound approximation with well chosen sutures, asepsis and psychic fortification. Early rising is defined as standing or walking in the first twenty-four postoperative hours. Patients may require encouragement and assistance in arising from bed as soon as their postoperative recovery permits. Careful expression of air from the peritoneal cavity at the time of peritoneal closure reduces reaction from this source when the patient arises.

The virtues of early rising are: (1) accelerated healing of tissues, (2) reduced disruption of the wound, (3) minimal thrombic and emboli formation, (4) less gastric and intestinal distention, (5) fewer bladder complications, (6) fewer enemas, (7) reduction in the amount of nursing care needed, (8) greater opportunity for self-help by the patient, (9) improvement of the patient's morale, (10) less asthenia, and (11) shortened hospital stay with resulting economy.[46]

Certain surgical situations or circumstances preclude early rising. Some of these conditions include: (1) fulminating peritonitis, (2) advanced cardiac disease, (3) pancreatitis, (4) hemorrhage not surgically controlled, (5) extensive phlebitis, (6) advanced cachexia and/or jaundice, and (7) large drains and/or temponades.[24]

Postoperative Complications. Complications that develop postoperatively generally reflect upon the care and foresight of the surgeon. It is his duty to anticipate the complications that may occur and circumvent them. Complications arise mainly from trauma which may be mechanical, infectious, chemical or psychic in nature.[1] The surgeon who operates with minimal trauma will in turn minimize subsequent complications. It is wiser to be adept at preventing complications than skilled in their treatment.

Trauma of mechanical origin accounts for most surgical complications. The more prevalent causative factors are failure to practice scrupulous hemostasis, exposing serosal surfaces and allowing them to become cool and dry, blunt dissection, mass ligation of tissue, indiscriminate crushing of blood vessels, rough handling of other tissues, tension upon visceral structures, prolonged mechanical retraction, allowing hollow viscera to become overdistended and strangulation instead of approximation in closing tissue layers. These surgical indiscretions give rise to shock, pain, distention, nausea, vomiting, ileus, thrombosis, embolus, urinary retention, etc. The surgeon must be gentle while he executes his task with dispatch.

Trauma of an infectious origin may arise at the site of surgery from whence it may be dissiminated elsewhere or it may be introduced into the surgical area and other sites from an external source. If infection is present, endeavor to limit its spread and microscopically identify by smear the causative organism. Frequently, smears from an apparently clean area may reveal organisms that bode harm unless promptly dealt with. In every case endeavor to combat a known causative organism not a group of unfavorable symptoms. Infection in the abdominal cavity when local calls for drainage; when not localized it probably defies drainage. It is necessary to mention the superior surgical results obtained at the Jackson Clinic by the local use of sulfanilamide and sulfathiazole. As much as 5 to 15 Gm. may be placed in grossly infected peritoneal spaces and frequently closed without drainage. It is also well to note the apparent catalytic effect of sulfathiazole and sulfadiazine when administered simultaneously. The sulfa drugs and penicillin are effective surgical handmaidens but they permit no surgical liberties.[14,47]

Trauma of chemical origin manifests itself mainly by changes in acid-base, nitrogen, water and electrolytic balance. In the body sodium ions make up the chief base and chloride ions are the chief acid radicals. Vomiting can and often does produce alkalosis and is relieved by adequate salt and water. Starvation, diabetes mellitus and loss of intestinal secretions may produce acidosis which calls for glucose, salt, water and at times insulin. Blood chemistry determinations are helpful in regulating these postoperative metabolic disorders.

Trauma of psychic origin includes all the mental changes that surgery and its attended rigors produce. This may vary from fear to a true psychosis. Avoid when possible operating persons who feel they "will not get well." Do not forget that

many psychotic persons subject themselves willingly to surgery and, unless wary, the surgeon may unknowingly "fix" such a psychotic abberation. Cutler has aptly stated that one cannot treat the body well unless one also treats the mind.

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THE PREOPERATIVE AND POSTOPERATIVE CARE IN MAJOR COLONIC SURGERY*

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THE surgeon who would operate upon the colon is confronted with several difficulties which make his work fraught with exceptional danger: (1) Most major surgery of the colon is performed for cancer with its concomitant tendency to debilitation. (2) The colon is filled with a large number of virulent bacteria. (3) The ramifications and interanastomoses of blood vessels supplying the colon are much less free than is the case with the stomach or small intestine. (4) A large proportion of colonic patients are elderly and in poor general condition; and (5) many colonic patients are partially or completely obstructed when first seen, particularly those with sigmoidal lesions.

All these factors add up to the result that many, if not most colonic patients, are subjected to radical and shocking surgery in the face of lowered resistance. Obviously, then, pre- and postoperative care must become equally important with a competent technic. It is the purpose of this paper to present a workable regimen which will yet be flexible enough to permit of modification according to individual circumstances.

PREOPERATIVE CARE

In Absence of Obstruction. In preparing an unobstructed patient for colonic surgery, several desiderata should be fulfilled: (1) The bowels should be empty; (2) peristalsis should be quieted at time of operation; (3) the cardiovascular condition should be as satisfactory as possible; (4) the patient should be well hydrated; and (5) body chemistry, particularly the protein, should be at normal levels.

It takes about five days to empty the bowel satisfactorily, and this is done last, following the patient's general workup or simultaneous with it. In addition to a thorough history and physical examination including, of course, sigmoidoscopy and biopsy if possible, *x*-ray studies are done. These consist of flat plate and barium enema followed by a double-contrast air-injection picture. Unless definitely indicated, barium is not administered by mouth, as this occasionally results in obstructing an otherwise unobstructed lesion. If the neoplasm is above the reach of the sigmoidoscope, the roentgenologist will often be able to give an exact localization, which is important in planning the operation. An estimate of the cardiac condition is made, including electrocardiographic readings when indicated. Blood studies are also done as indicated, but always include the following:

1. Complete count
2. Cephalin flocculation test
3. Blood chlorides
4. Blood urea
5. Blood creatinine
6. Blood sugar

(3–6: mg. per 100 cc.)

7. Total protein (A-G ratio)
8. Hgb.

(7–8: Gm. per 100 cc.)

9. Hematocrit

These findings, together with other data, are recorded on a "Colon Sheet."

It will almost invariably be found that the total protein is low and strenuous efforts are made in the preoperative period to obtain a level of 6.5 Gm. per 100 cc. or higher.

If this level cannot be reached, it is not considered an absolute bar to operation

* From the Surgical Service of Dr. Ottokar Tenopyr, Kings County Hospital, Brooklyn, N. Y.

but it is believed that the risk is definitely increased. Means adopted for increasing the plasma protein level are: (1) High protein intake by mouth, particularly beefsteak if obtainable; (2) enzymatic hydrolysate of casein and pancreas* 1,000 cc. intravenously daily; (3) oral amigen 2 oz. three times a day usually in grapefruit juice. It is found that although many patients complain about the flavor of oral amigen, they will take it when the necessity is explained; (4) plasma or transfusion as indicated, considering also the degree of anemia.

Other chemical defects are handled in the usual manner; diabetes is treated as such but with insulin rather than by reduction of carbohydrates. If the glucose imbalance is marked, the treatment should be under the control of a competent internist. Chlorides are easily raised by raising the salt intake. If the patient is vomiting, normal saline solution is administered intravenously, but this is ordinarily not the case with unobstructed patients.

To accomplish the objects discussed above, orders are written approximately as follows, but with due regard to the exigencies of the individual case:

1. High protein, high carbohydrate, low residue diet.
2. Multivitamin tablets 2 t.i.d.
3. Type blood (and cross-match 1,000 cc.)
4. Amigen 1,000 cc. intravenously daily.
5. Amigen oral—2 oz. in fruit juice t.i.d.
6. Total plasma protein daily (see discussion of Van Slyke test later in this paper.)
7. Electrocardiogram (if indicated).
8. Transfuse (if indicated).
9. Sulfasuxidine 2 Gm. initial dose followed by 1 Gm. every four hours.
10. Insulin, digitalis, etc., (if and as indicated.)

*The product used in our cases is Amigen, manufactured by Mead Johnson & Co.

Next, starting five days preoperatively, the following orders are written:

First day—Licorice powder drams 2 at night.
Second day—6 A.M. magnesium sulfate 50 per cent—1 oz.
Third day—6 A.M. magnesium sulfate 50 per cent—½ oz.
Fourth day—6 A.M. magnesium sulfate 50 per cent—½ oz.
Fifth day—(day before operation)
6 A.M. castor oil—1½ oz.
6 P.M. paregoric drams 1
6 P.M. 1 pint tap-water enemas until clear

Morning of operation:

1. 1,000 cc. saline intravenously before going to operating room.
2. Catheterize and leave catheter in place.
3. Levine tube in stomach.
4. Preoperative medication.
5. Transfusion of citrated blood.

Preoperative Care When Patient Is Obstructed. The presence of partial or complete obstruction profoundly alters the situation. Not only does surgery in the presence of obstruction offer technical difficulties in the shape of a distended bowel and sometimes a vomiting patient, but also the bowel proximal to the point of obstruction contains organisms of vicious virulence and the walls of the gut are greatly devitalized so that needle holes do not close tightly and suture lines do not hold so well. It is, therefore, of the greatest importance to relieve the obstruction by medical means if at all possible prior to surgery. To this end, Wangensteen suction is instituted from above and high colonic irrigations two to three times daily from below. In addition, sulfadiazine is administered intravenously with the idea that if some of the obstructing mass is inflammatory, as it ordinarily is, the exhibition of potent chemotherapy may aid in reducing its size. Very occasionally, an obstructing lesion in the upper rectum may be by-

passed by a small catheter inserted under direct vision through a proctoscope.

It is quite astonishing how often a markedly distended abdomen will be decompressed by these maneuvers. It should be borne in mind, however, that the Wangensteen suction is intended only to control vomiting and small intestinal distention; neither it nor the Miller-Abbott tube will ordinarily be effective in reducing a distended colon.

If it has been possible to overcome the obstruction by the means outlined above, the bowel is then emptied as well as possible from below (the use of cathartics is rather dangerous here and should be subjected to careful judgment). It is usually the procedure of choice to perform a two-stage operation in these patients, the first stage being one form or other of colostomy; and in any case, primary anastomosis of the left colon is not done in any patient even slightly obstructed.

The body chemistry is investigated and corrected as nearly as possible in the manner described for unobstructed patients. Sometimes, however, a surgical decompression must be done too soon to permit an ideal workup.

POSTOPERATIVE CARE

Having performed an extensive operation—and colonic surgery for carcinoma should be extensive—the next problem is to keep the patient alive through three primary dangers: shock, pneumonia and peritonitis, of which the last is fortunately becoming uncommon.

At the time of operation, 10 Gm. of crystalline sulfanilamide are often placed about the suture line or area of possible contamination. Sulfathiazole is not used because of its tendency to cake. A transfusion of at least 500 cc. and sometimes 1,000 cc. is given during and following the operation.

Shock is to be detected and treated in its infancy—long before blood pressure drop ensues. To this end, hematocrit readings are checked frequently by the Van Slyke copper sulfate method and plasma is given when indicated. Continuously rising hematocrit indicates imminent shock and continuously dropping hematocrit indicates hemorrhage.

Pneumonia is to be anticipated and combatted by the use of penicillin, early ambulation, frequent changes of position and proper aeration of lungs.

Peritonitis is combatted by careful operative technic and the use of sulfonamides, with emphasis upon the former. It is believed that the sulfonamides are more effective than penicillin for colonic bacteria.

Fluid and electrolyte balance are also to be maintained.

These desiderata are translated into the following more or less routine orders which are, of course, modified to suit the individual case:

1. Fluids, at least 3,000 cc. in twenty-four hours, divided thus:
   Amigen: 1,000 cc.
   Glucose 5 per cent in normal saline: 1,000 cc.
   Glucose 10 per cent in water: 1,000 cc.
2. Vomitus or Wangensteen drainage to be measured and this amount added to the requirement of saline solution.
3. Apply Wangensteen suction to Levine tube which is already in place.
4. Oxygen tent. (This is not routine but is utilized at the slightest provocation.)
5. Turn patient from side to side every few minutes.
6. Out of bed in wheelchair for fifteen minutes within twenty-four to forty-eight hours if possible. (Judgment as to this rests on condition of patient, method of suturing wound, etc.)
7. Sodium sulfadiazine intravenously—6 to 7 Gm. in twenty-four hours until peristalsis returns and patient is accepting food by mouth. (After this time sulfasuxidine may or may not be given.)
8. Penicillin 50,000 units intramuscularly stat—then 30,000 units every three hours (until danger of pneumonia is considered slight).

9. Daily blood sulfa level (to be maintained between eight and 12 mg.)

10. Morphine sulfate gr. ⅙ every four hours p.r.n.; (as little as possible.)

11. Van Slyke test every two to four hours on the first day, then daily.

12. Moderate Trendelenburg for six hours (for pulmonary drainage) then gradually flat and changed to semi-Fowler with pillow under each axilla.

13. No enema for five to six days, and then only in small amounts.

14. Insert rectal tube for 3 inches only.

In cases of clamped colostomies, the clamp is customarily removed from the proximal (working) opening in forty-eight to seventy-two hours, but there is no objection to removing it in thirty-six hours if distention or other symptoms make it desirable.* If a distal opening is present and clamped, as in a first stage Lahey operation, this clamp remains in place until it falls off, which is usually in about a week.

PRIMARY ANASTOMOSIS

In the right colon, primary anastomosis is done routinely, except in cases of emergency in which it is imperative to stop the operation as soon as possible. Ascending colectomy is accompanied by ileotransverse colostomy and the suture line is ordinarily, though not invariably, decompressed by either a proximal ileostomy after the method of Witzel or a Miller-Abbott tube inserted a few days in advance in order to permit it to reach the ileum.

In the left colon, the dangers of primary anastomosis are not inconsiderable, since there is likely to be at least some degree of obstruction and the resultant devitalizing effect on the bowel wall may permit leakage through suture holes if not actually through the line of anastomosis. If, however, obstruction can be completely and definitely overcome for a period of two weeks by means of a proximal colostomy, primary anastomosis is often feasible.

* With the use of a Daniel clamp[3] decompression is attained immediately.

CHEMOTHERAPY

Penicillin is a powerful aid against pneumonia, but is inferior to the sulfonamides in combatting colonic bacteria. For this reason, penicillin is administered only after operation and the sulfonamides both before and after surgery.

As soon as peristalsis has started and stools are being passed, suction is gradually discontinued and feedings by mouth are begun. This is usually in about three to four days. At this time also intravenous sulfadiazine may be discontinued and replaced, if desired, by sulfasuxidine orally.

VAN SLYKE TEST

This test, known also as the copper sulfate test, is one of the most useful laboratory procedures yet devised. By means of a comparatively simple maneuver performed in five minutes in the ward by the intern, the following information is obtained: plasma protein, hematocrit and hemoglobin.[11]

ANESTHETIC

It is believed that intrathecal procaine in competent hands is the anesthetic of choice. However, in the absence of a trained anesthesiologist, who can both administer and take complete charge of the progress of anesthesia, general anesthetics are resorted to. For critically ill patients who are being subjected to emergency surgery for obstruction, local anesthesia is often utilized. In this regard, it has been our experience that if the line of skin incision is injected intradermally with 1 per cent novocaine, a minimal quantity of ¼ per cent novocaine will suffice for the remainder of the operation. In order to lessen the number of external skin punctures, the skin may be infiltrated by inserting the needle subcutaneously at one end of the proposed line of incision and creating a series of wheals from the under surface.

EVISCERATION

This is fortunately a rare complication of colonic surgery. It is believed that while

the plasma protein and especially the plasma albumin level have a distinct bearing on the problem, there must be other factors of importance, since occasionally a non-infected wound breakdown will be seen with normal protein levels. When a wound opens down to but not through the peritoneum, it may safely be packed with vaseline gauze; but when actual evisceration take place, through-and-through sutures with heavy silk, silkworm gut or steel alloy wire should be inserted.

COLON SHEET

Dr. Ross _____ Patient's Name

Date										
Blood										
Chloride										
Urea										
Creatinine										
Sugar										
Sulfa level										
Tot. Prot.										
Alb.										
Glob.										
Hgb.										
Hematocrit										
Cephalin floc.										
Urinary sugar										
Digitalis reserve										
Penicillin 24 hr. tot.										
24 hr. tot.										
Fluid Intake										
Fluid Output										
24 hr. tot.										
Amigen										
Glucose 5%										
Glucose and saline										
Normal saline										
Tot. NaCl as saline										
Citrated blood										
Plasma										

EMERGENCY COLOSTOMY

Colostomy as an emergency measure has so many disadvantages that it is to be avoided if at all possible. To this end it is considered that several hours may well be spent before resorting to emergency colostomy in attempts to decompress the large bowel by high colonic irrigations. This time may be utilized to advantage in giving a transfusion and otherwise attempting to improve the patient's condition. In this connection also it may be noted that intravenous sulfonamides have a distinct tendency to aid in relieving an acute obstruction, although twenty-four hours is usually necessary for the action to show itself. The explanation of this phenomenon undoubtedly lies in the fact that the obstructing carcinoma is ordinarily accompanied by considerable inflammatory swelling. It is the function of the sulfonamides to decrease this swelling, and occasionally such decrease will result in the passage of sufficient flatus or stool to accomplish a satisfactory decompression, thus changing an emergency procedure into an elective one.

MISCELLANEOUS CONSIDERATIONS

For convenience in keeping track of the various laboratory tests and other data routinely used in these cases, a "Colon Sheet" has been devised and found useful.

In administering intravenous amino acid preparations such as Amigen, it is important to remember that their incompatibilities are many. In fact, it is probably best to administer these very useful substances

alone and to avoid the temptation to add blood plasma, sulfonamides or other preparations to the infusion containing the hydrolysate.

SUMMARY

1. A basic regimen has been presented for the pre- and postoperative care of colonic surgery.

2. Views are presented on the subjects of primary anastomosis of the left colon, chemotherapy, the Van Slyke test, choice of anesthetic, postoperative evisceration and emergency colostomy.

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Cryptitis is a state of inflammation in a crypt of Morgagni. This will usually precede a papillitis. Papillitis is an inflammatory reaction of a papilla, usually associated with edema and hypertrophy.

From "Ambulatory Proctology" by Alfred J. Cantor (Paul B. Hoeber, Inc.)

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2. Lift off SAFTI-CAP

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Just as the insoluble barium salts have made x-ray exploration of the alimentary tract a practical procedure, so has Lipiodol made possible roentgen probing of other radiolucent systems and body spaces. Containing 40% iodine firmly bound to poppy-seed oil, Lipiodol possesses a high degree of radiopacity. When instilled into body cavities, it sharply delineates their configuration on the x-ray film. Its bold shadow readily demonstrates distortions and impingements upon normal structures—breaks in continuity caused by pathologic as well as traumatic processes.

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1. Bunn, P. A.: in Conferences on Therapy: New York State J. Med. 46:527 (March 1) 1946.
2. György, P.; Evans, K. W.; Rose, E. K.; Perlingiero, J. G., and Elias, W. F.: Pennsylvania M. J. 49:409 (Jan.) 1946.

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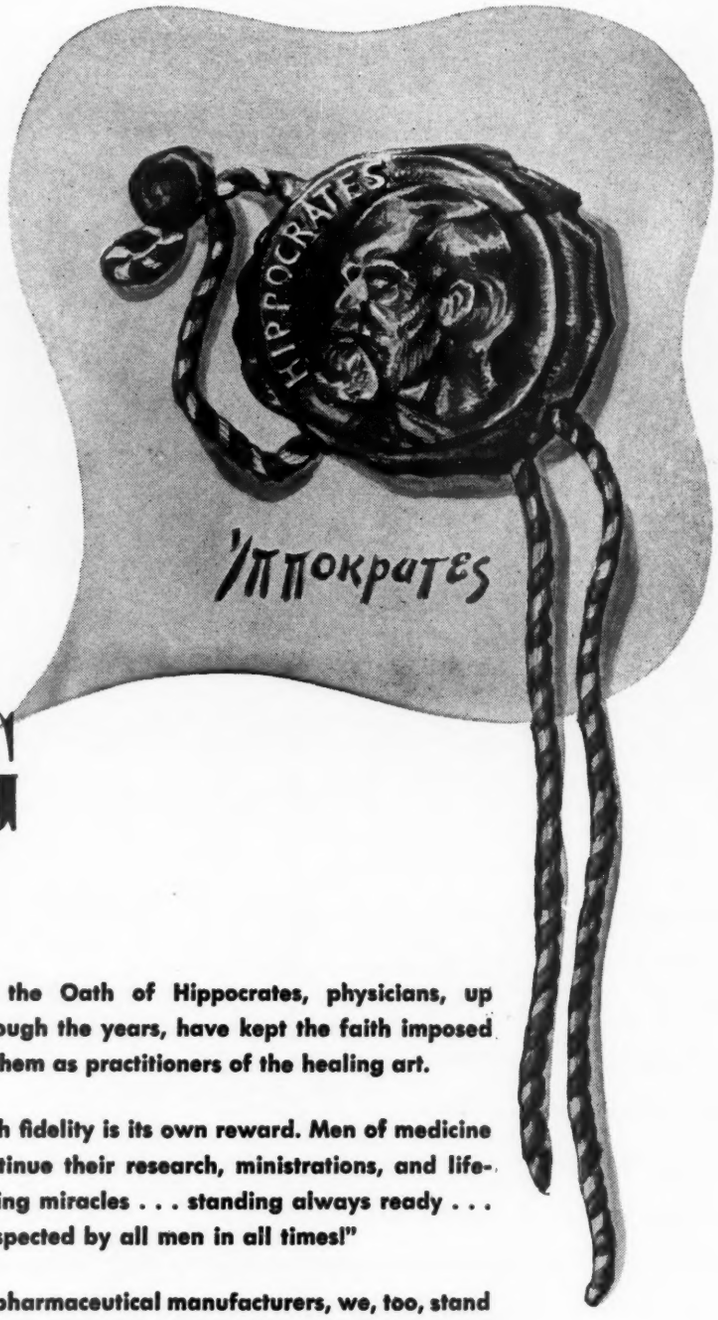

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By the Oath of Hippocrates, physicians, up through the years, have kept the faith imposed in them as practitioners of the healing art.

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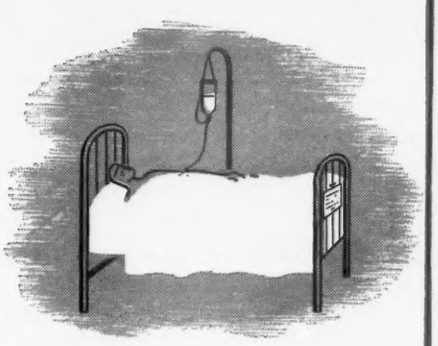
"... the protein deficient individual is a poor operative risk."
Lund and Levenson: J.A.M.A. 128:95, 1945

"When time is available to improve protein nutrition before surgery and when this time is used efficiently for this purpose, the reduction in postoperative shock and other complications is impressive."

Editorial: Surg., Gynec. & Obst. 83:259, 1946



POSTOPERATIVE

REPARATION

"... the patient maintained in positive nitrogen balance recovers from major surgery more rapidly than does the patient who is not in nitrogen equilibrium."

Koop: Geriatrics 1:269, 1946

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FOR PROTEIN DEFICIENCY

Preparative: To improve and protect the nutritional status of the severely malnourished or critically ill patient . . . as fortification against the shock of major surgery.

Reparative: To provide, in ample quantity, the amino acids essential to tissue repair . . . to hasten healing and shorten convalescence.

Parenamine is a 15 per cent sterile solution of all the amino acids known to be essential for humans . . . derived by acid hydrolysis from casein and fortified with *dl*-tryptophane.

For use whenever dietary measures are inadequate for correction and maintenance of positive nitrogen balance . . . to replenish depleted body protein stores. *Particularly indicated* in pre- and postoperative management, extensive burns, gastro-intestinal obstruction, etc.

Supplied in 100 cc. rubber-capped bottles.

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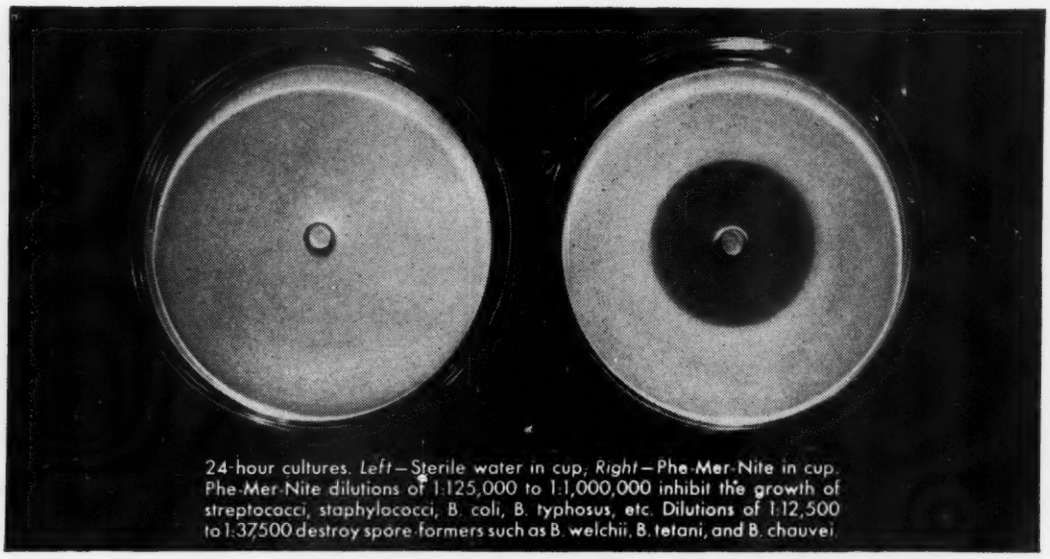

24-hour cultures. *Left*—Sterile water in cup; *Right*—Phe-Mer-Nite in cup. Phe-Mer-Nite dilutions of 1:125,000 to 1:1,000,000 inhibit the growth of streptococci, staphylococci, B. coli, B. typhosus, etc. Dilutions of 1:12,500 to 1:37,500 destroy spore-formers such as B. welchii, B. tetani, and B. chauvei.

High Bactericidal Power— Minimal Tissue Toxicity

Phe-Mer-Nite is powerfully bactericidal and fungicidal, yet nonirritating and harmless to tissue in the recommended dilutions. It does not hemolyze red blood cells, does not precipitate tissue proteins, has no offensive odor, does not stain. It maintains its bactericidal and fungicidal powers in the presence of blood, pus or exudates. It is stable to light and high temperatures; long standing does not reduce its potency.

Both on intact surfaces and in open wounds, Phe-Mer-Nite fulfills every need for dependable antisepsis. It may be applied to burns or other lesions requiring antisepsis, and is useful as a douche, nasal spray, gargle, and for the sterilization of instruments and rubber gloves. Phe-Mer-Nite is particularly useful in the treatment of ringworm infestations.

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Phe-Mer-Nite, a brand of phenylmercuric nitrate, is an organic salt of mercury of low toxicity and high germicidal power.

Available as a tincture, solution, in cholesterinized or greaseless base, in throat tablets, and in foot powder.

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SE Mco

Breakfast and the Daily Protein Need

The significance of breakfast in the satisfaction of nutritional requirements has been emphasized in many quarters in the recent past. Breakfast serves to replenish many nutrient stores depleted during the long fast from the previous evening meal, and provides the organism with caloric food energy needed for maximum efficiency during the morning hours. Hence nutrition authorities advise that breakfast should supply from one-fourth to one-third of the daily caloric and nutrient needs.

The morning meal should provide, among other things, its share of the daily protein requirement, since the protein needs must be met daily for proper growth of children and for good nutritional health of adults. In a basic breakfast so widely recommended—fruit, cereal, milk, bread and butter—the protein contribution is significantly high—20.7 Gm., or about 29 per cent of the adult requirement. Not a small amount of this protein is provided by the average serving of cereal (ready to eat or to be cooked), milk and sugar—fully 10 per cent of the adult daily protein need. Thus an important protein contribution is made by the basic breakfast, of which cereals are an integral and universally recommended component.

This average cereal serving also provides B complex vitamins, caloric food energy, and important minerals. Its mixture of proteins is of high biologic value, applicable for the satisfaction of growth and maintenance requirements. Note from the table of composite averages the contribution made by the cereal serving—1 ounce of cereal (whole grain, enriched, or restored to whole-grain values of thiamine, niacin, and iron), 4 ounces of milk, and 1 teaspoonful of sugar—and by the basic breakfast.

	Nutrition Composition of The Basic Breakfast*	Average represented by: cereal, 1 oz.; whole milk, 4 oz.; sugar, 1 teaspoonful
Calories	611	202
Protein	20.7 Gm.	7.1 Gm.
Fat	19.0 Gm.	5.0 Gm.
Carbohydrate	89.4 Gm.	33.0 Gm.
Calcium	0.465 Gm.	0.156 Gm.
Iron	3.0 mg.	1.6 mg.
Vitamin A	1074 I.U.	193 I.U.
Thiamine	0.52 mg.	0.17 mg.
Riboflavin	0.87 mg.	0.24 mg.
Niacin	2.3 mg.	1.4 mg.
Ascorbic Acid	64.8 mg.	

*Orange juice, 4 oz.; cereal, 1 oz.; milk, 4 oz.; sugar, 1 tsp.; bread (enriched, white), 2 slices; butter, 1 tsp. (5 Gm.); milk, 8 oz.

The presence of this seal indicates that all nutritional statements in this advertisement have been found acceptable by the Council on Foods and Nutrition of the American Medical Association.

CEREAL INSTITUTE, INC.

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HEPTUNA—A potent and effective approach in the management of hypochromic anemia with its multiple nutritional and other systemic manifestations.

EACH CAPSULE CONTAINS:

Ferrous Sulfate U.S.P.	4½ Grains
Vitamin A (Fish-Liver Oil)	5,000 U.S.P. Units
Vitamin D (Tuna-Liver Oil)	500 U.S.P. Units
Vitamin B_1 (Thiamine Hydrochloride)	2 mg.
Vitamin B_2 (Riboflavin)	2 mg.
Vitamin B_6 (Pyridoxine Hydrochloride)	0.1 mg.
Calcium Pantothenate	0.333 mg.
Niacinamide	10 mg.

Together with a Liver Concentrate (Vitamin fraction) derived from 6.5 Gm. fresh liver and dried yeast U.S.P. Not intended for use in the treatment of pernicious anemia.

Heptuna

For the speedy correction of the anemia syndrome and its associated multiple nutritional deficiencies, iron alone is usually inadequate. All the lacking essential nutrients must be supplied, by both diet and appropriate medication.

Supplied in boxes of 50 and 100 capsules

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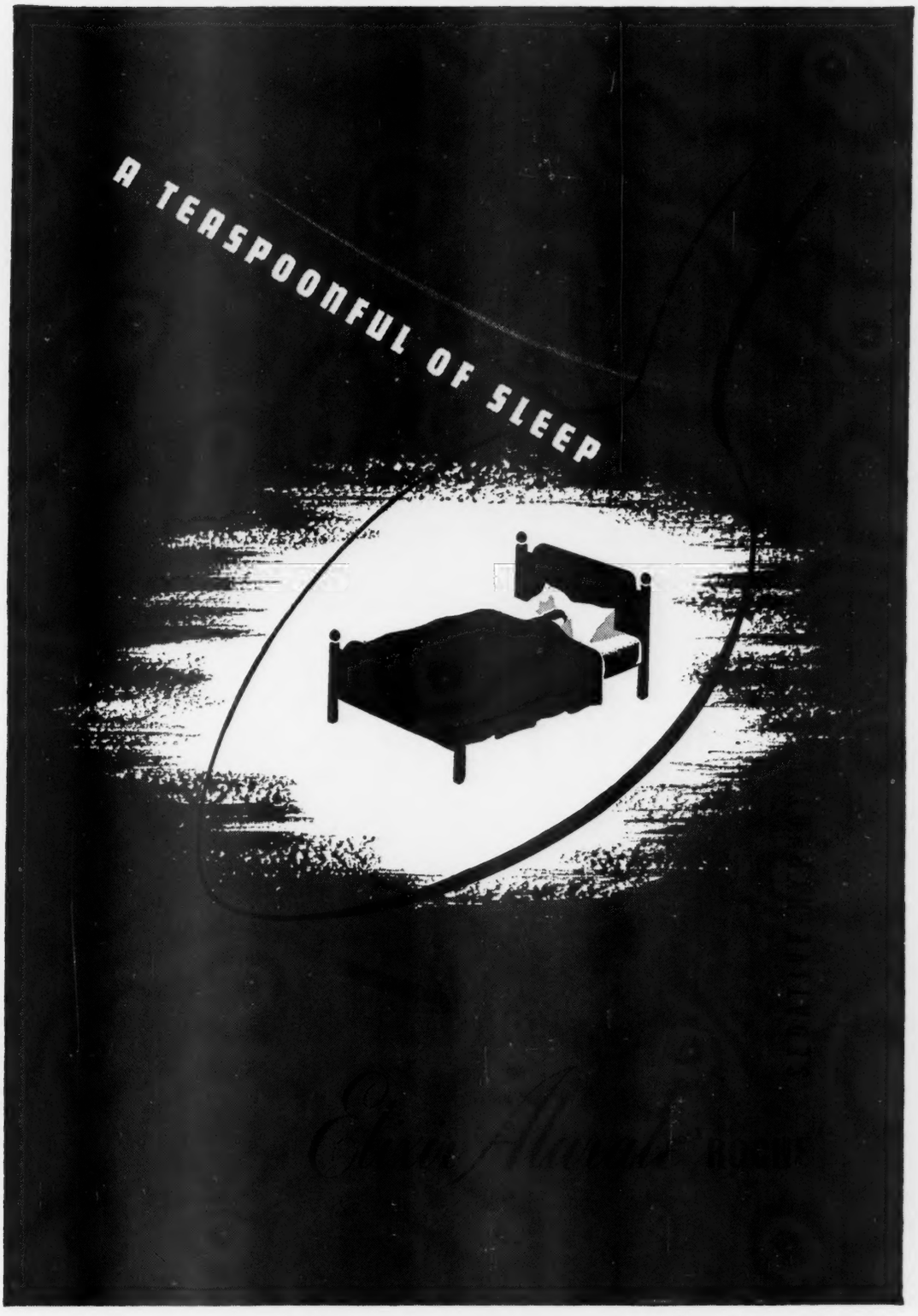

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PROCTOLOGY AND GASTRO-ENTEROLOGY

A combined course comprising attendance at clinics and lectures; instruction in examination, diagnosis and treatment; witnessing operations; ward rounds; demonstrations of cases; pathology; radiology; anatomy; operative proctology on the cadaver.

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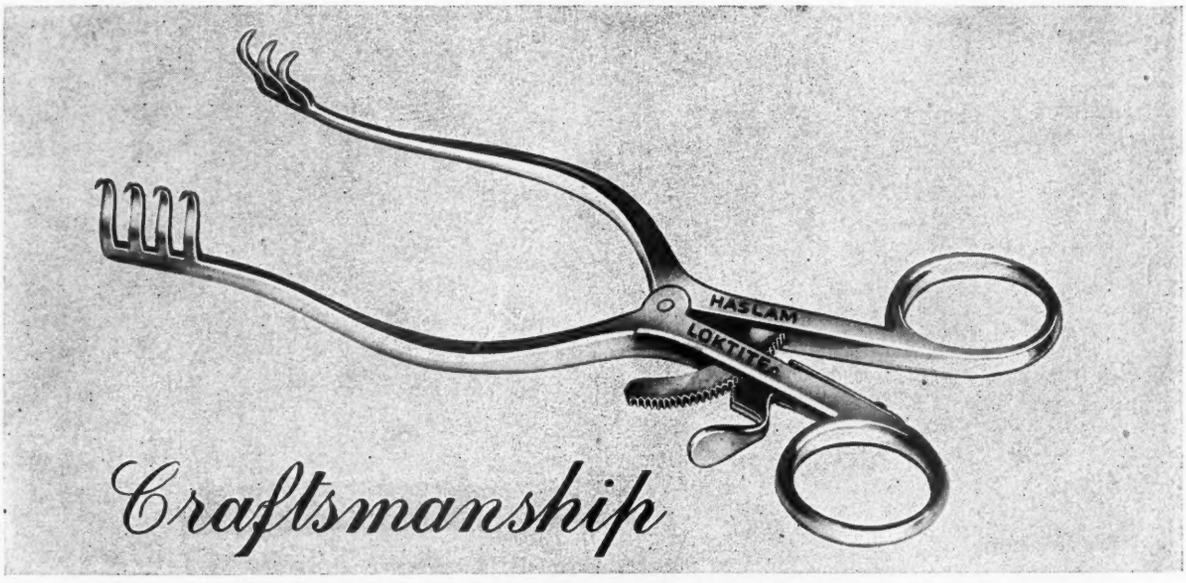

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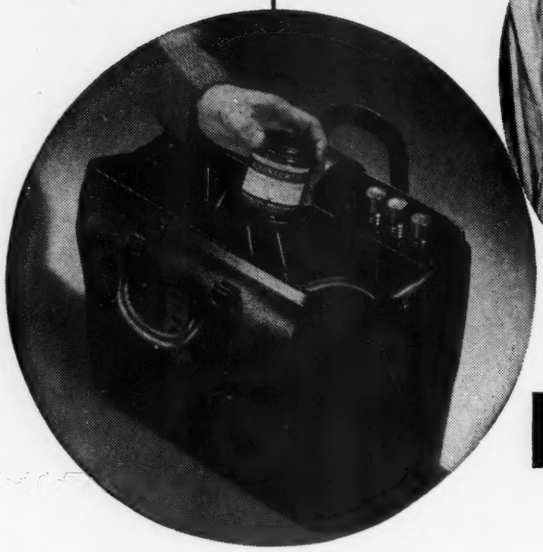

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Whatever the surgical application, Allantomide is of established usefulness in preventing delay in the normal healing process, aiding revascularization of granulation tissue, and guarding against re-infection.

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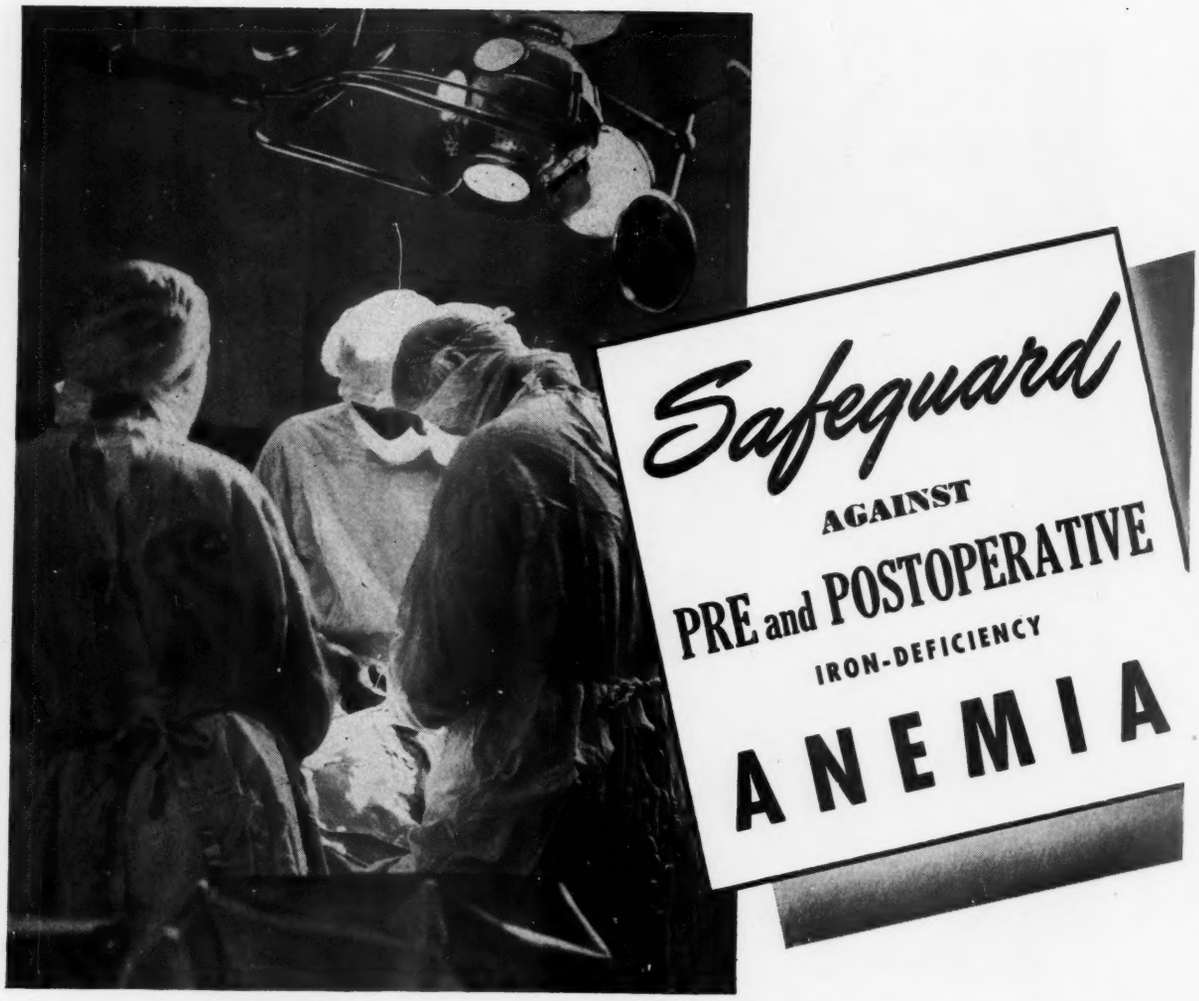

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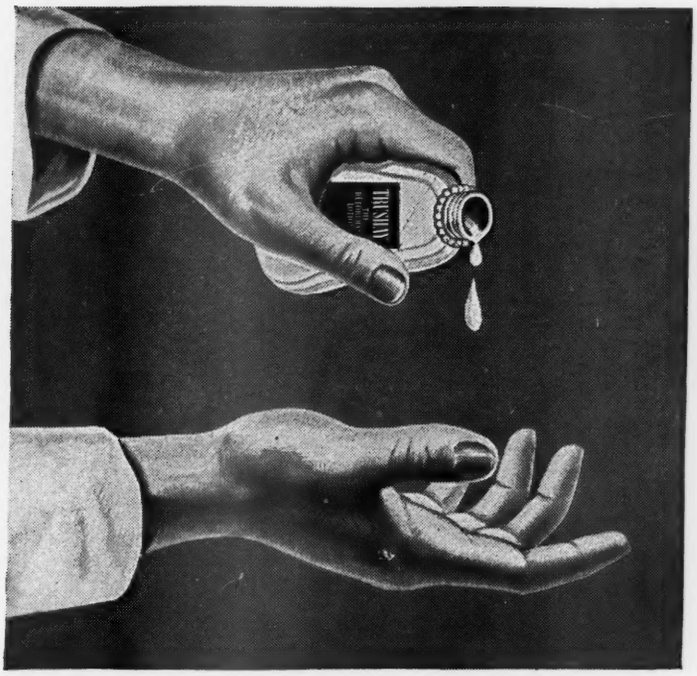

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But if, after washing, a cream or lotion is applied to the hands to prevent redness or roughness, some of the hygienic value of soap and water cleansing undoubtedly is lost.

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TRUSHAY helps prevent depletion of the skin's natural lubricant . . . aids in keeping the dermal tissue normal and unbroken.

And since TRUSHAY is applied *before* washing, hands can be soft, smooth and well-groomed and still meet the requirements of modern hygiene.

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The "Beforehand" Lotion

A Product of Bristol-Myers, 19 NF W. 50th St., New York 20, N. Y.

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"STOKED BY THE FIRES OF PERDITION"

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Its necessity in the prevention of Goiter and its usefulness in the treatment of respiratory conditions are important chapters in its service record.
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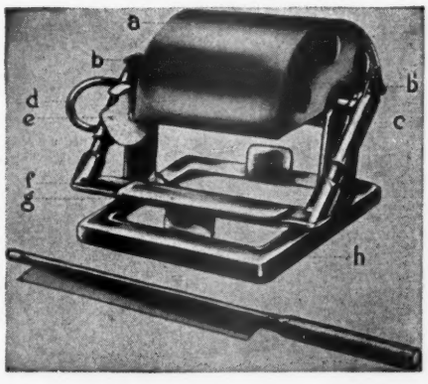

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VAGINAL JELLY

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The American Journal of Surgery

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